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DISEASES OF THE NERVOUS SYSTEM

DISEASES OF THE NERVOUS SYSTEM

BY

H. CAMPBELL THOMSON
M.D. (Lond.), F.R.C.P.
Physician to the Department for Diseases of the Nervous System, and Lecturer on Neurology, Middlesex Hospital: Physician to the Hospital for Epilepsy and Paralysis, Maida Vale; formerly Dean of the Middlesex Hospital Medical School

Third Edition, Revised

WITH 11 COLOUR AND 12 BLACK-AND-WHITE PLATES AND 120 FIGURES IN THE TEXT

New York
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1921

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TO

SIR JAMES KINGSTON FOWLER, K.C.V.O., C.M.G.

IN GRATEFUL APPRECIATION OF
HIS TEACHING AND COUNSEL

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PREFACE TO THE THIRD EDITION

In preparing this manual for a Third Edition, I have endeavoured to incorporate such new matter as is essential without materially increasing the size of the book.

The knowledge obtained by the researches of Henry Head, Gordon Holmes, Sir W. Lister, Riddoch, and others, during the War, especially in connection with the effects of injuries to the spinal cord, the theory of aphasia, and the functions of the cerebral cortex regarding sensation and vision, has necessitated the addition of not a little that is new and the modification of much that is old.

Among other subjects now introduced into the book is Encephalitis Lethargica. The relation between the Neuroses and the internal secretions has been emphasized, and a fuller description given of the Autonomic System.

The literature of the Psychoneuroses has become so extensive that it is difficult to deal with the subject in the space at my disposal, and in largely rewriting and rearranging this section I have contented myself with an endeavour to present the more salient points in such perspective as will enable the student to pursue his investigations in any branches which he may desire to explore further. Arguments as to the relative values of the different schools of psychotherapy I have thought it well to avoid.

I am greatly indebted to Prof. James McIntosh, Dr. George Riddoch, Dr. Percy Whittick, and to many other friends and colleagues, for the advice and assistance which they have kindly given me.

H. C. T.

34, Queen Anne Street, W.1.
June, 1921.

PREFACE TO THE FIRST EDITION

In attempting to marshal the main facts of neurology in a concise and yet, I hope, readable manner, it has been my constant aim to bear in mind the principal difficulties which are apt to beset the student.

The experience on which this book is founded has been obtained for the most part at the Hospital for Nervous Diseases, Maida Vale, and at the clinics for nervous diseases which I have for some years past held at the Middlesex Hospital. To my colleagues at both these hospitals I express my hearty thanks for the many opportunities they have kindly given me of observing cases under their care.

To the courtesy and generosity of Professor Pierre Marie in supplying me with photographs for illustrations I owe a special debt of gratitude, as also I do to Mr. Bland-Sutton for the loan of blocks, and to others who have kindly allowed me to reproduce illustrations, the sources of all of which, I trust, are duly acknowledged.

For taking photographs of my cases I have chiefly to thank Mr. Mann, and for the taking of the instantaneous photographs reproduced in four of the plates I am indebted to Mr. Charles Urban.

Finally, my best thanks are due to the publishers for the valuable assistance they have given me throughout the preparation of the book.

H. CAMPBELL THOMSON.

April, 1908.

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DISEASES OF THE NERVOUS SYSTEM

SECTION I.—INTRODUCTORY

CHAPTER I

GENERAL STRUCTURE OF THE NERVOUS SYSTEM

THE nervous system is composed of nerve cells surrounded and supported by connective tissue. A **nerve cell** consists of a cell body and its processes. The processes which conduct impulses away from the cell body are called **axons**, while those which transmit impulses towards the cell body are known as **dendrons,** and the cell body together with all its branches constitutes the **neurone** of Waldeyer. The neurone is therefore a cell which has become specially differentiated to perform the function of conduction (Fig. 1.)

The cell body, as studied by Nissl's method of staining with aniline dyes, is principally made up of two substances which are qualified as chromatic and achromatic according to their staining properties.

The **chromatic substance** is arranged in cube-shaped patterns known as Nissl bodies, which are distributed in a fairly regular manner throughout the cell body. Although this tessellated appearance is probably largely due to the hardening reagents used in the preparation of the specimens, since it does not appear in cells that are stained during life, it is nevertheless an important guide for comparative purposes in estimating the changes that have taken place in the cell bodies as the result of disease. There is some uncertainty as to the function this chromatophile substance fulfils, but it is generally thought to be connected with the nutrition and energy of the cell. It undergoes various changes as the result of disease, among the more common being loss of substance (chromatolysis) or a uniform staining in which all definition of the chromatophile granules is obscured.

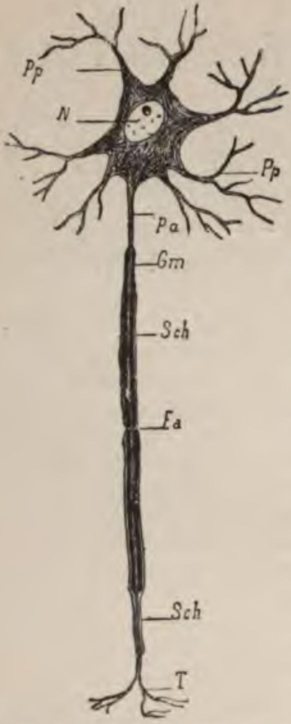


Fig. 1. — Diagrammatic representation of a neurone. (*Morat.*)

N, nucleus of the nerve cell; *Pp*, protoplasmic prolongations; *Pa*, axon, or axis cylinder; *Gm*, sheath of myelin; *Sch*, sheath of Schwann; *Ea*, annular node; *T*, terminal ramifications of the axis cylinder.

The unstainable or **achromatic substance** consists of a delicate network of fibres which appear to connect the axons with the dendrons, and is, therefore, presumably closely connected with the conduction of nerve impulses.

There is a nucleus generally situated near the middle of the cell body, and inside that a nucleolus which usually shows up well by reason of its darker staining. Pigment also may often be seen in the cell body, but the significance of its presence is uncertain.

The **axon,** as a rule, is single and of greater length than the dendron, and, as already mentioned, it generally conveys impulses away from the cell body and gives off branches, known as collaterals, along its course. Shortly after leaving the cell body the axon becomes encased successively with a sheath of myelin and a neurilemma or sheath of Schwann, both of which it discards a little before it breaks up into its terminal branches.

Such is a typical neurone, but, of course, the size and shape of the cell bodies and the length and thickness of their branches differ very greatly, and there are some neurones whose axis cylinders do not leave the grey matter, and do not in those circumstances acquire a medullated sheath.

According to the **neurone theory** every neurone is a separate anatomical unit capable of living and dying independently of every other neurone, and the processes of one have no physiological connection with those of another, i.e. the branches are contiguous but not continuous, just as the branches of trees, however intimately entwined with those of others, depend for their vitality upon the integrity of their own stems and roots.

It thus comes about that the nervous system is made up of an infinite number of neurones, bound up by connective tissue into various bundles and tracts according to the different kinds of impulses they conduct. There are, for example, special bundles for motor impulses — the motor tracts—and others for sensory impulses — the sensory tracts—and so on; but the ultimate elements making up all these bundles are simple neurones.

Doubt has occasionally been cast upon the correctness of the neurone theory, by which every neurone is supposed to have a complete individuality. Possibly it may not hold good in all the lower forms of animal life, but in the higher forms and in man the distribution of nerve degeneration, which is mainly limited by the termination of cell branches of particular tracts, and the embryological evidence of the independent development of different units, are both strongly in favour of its truth.

The mechanism by which impulses pass from one neurone to another is not accurately known. It has been thought that the terminal filaments of the neurones, upon which little buds or excrescences can often be seen, are capable of retraction and protrusion in an amœboid manner, thereby linking up or breaking connections, as the case may be; but this is at present largely a matter of conjecture.

A clear conception of the constitution of the nervous system as made up of collections of neurones is an important aid to the understanding of all nervous diseases, for it enables us to foretell to a large extent the course and limits of degenerations and to understand how certain tracts may be affected to the exclusion of others.

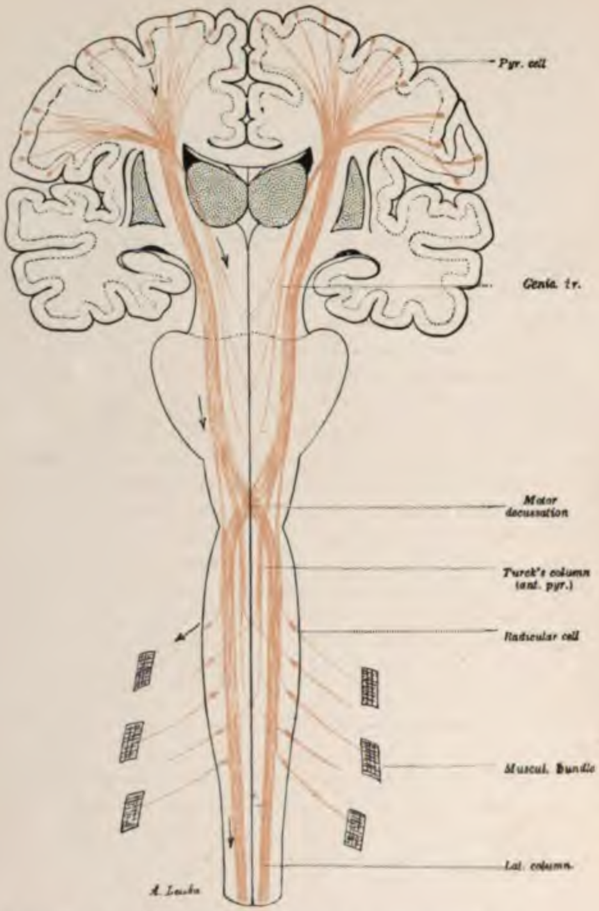


PLATE 1.—Motor field with its two principal orders of projection fibres. (*Morat.*)

CHAPTER II

GENERAL CLASSIFICATION OF NEURONES

THE neurones may be divided into the following groups, viz. :—

1. Projection neurones.
2. Association neurones.
3. Commissural neurones.

The **projection neurones** connect the brain with the spinal cord, and are divided again into descending and ascending groups according as they conduct impulses from the brain downwards or from the cord upwards. Examples of the down-going neurones are to be found in the crossed pyramidal tracts, the cell bodies of which are situated in the cerebral cortex, and of the up-going neurones in the long fibres of the posterior columns and in those of the cerebellar tracts. (Plates 1 and 3.)

The **association neurones** link one portion of the cortex of the brain with another, and in the spinal cord also there are fibres of different lengths which join up various segments. (*See* Fig. 8, p. 55.)

The **commissural neurones** connect the two segments of the brain and the two halves of the cord with each other, e.g. the fibres of the corpus callosum which bridge the space between the two hemispheres of the brain, and the fibres forming the commissure which unites the two halves of the spinal cord.

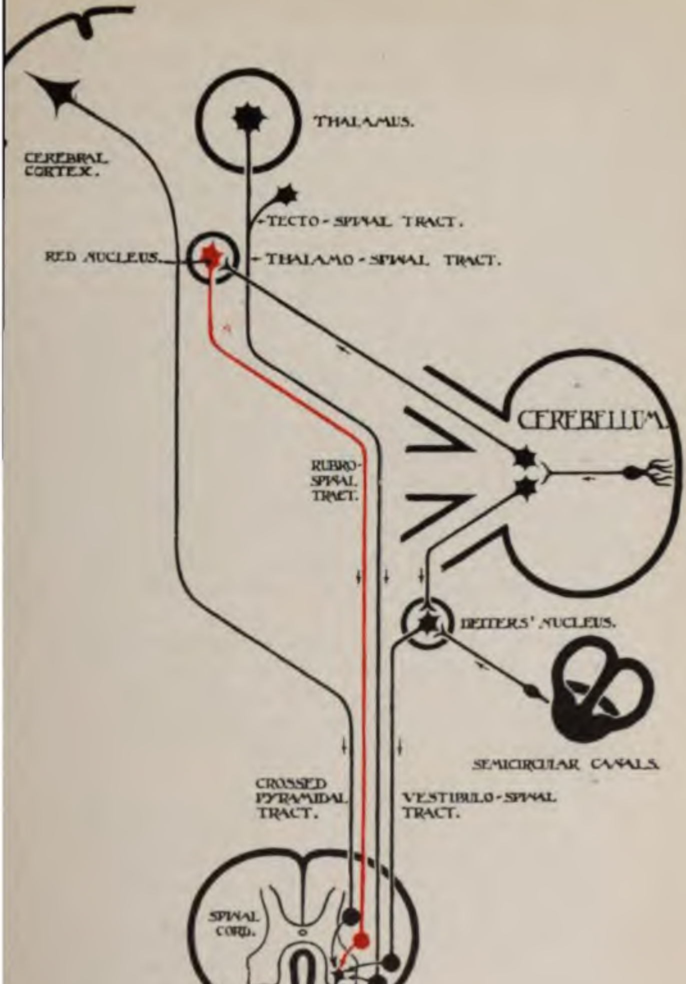
DESCENDING PROJECTION FIBRES

The principal descending projection fibres, or **upper motor neurones** as they are called, are those which make up the motor tract down which pass the impulses that give rise to voluntary movements. The cell bodies of these upper motor neurones are situated in the motor area of the cerebral cortex, and while their short branches (dendrons) interlace with others in all directions, their long ones (axons) converge to form the main motor tract. After coming off from their cell bodies in the cerebral cortex these fibres pass successively through the corona radiata, internal capsule, crus cerebri, pons, and medulla. At the level of the crus and the pons some of the fibres leave the main body, and, after decussating with their fellows of the opposite side, terminate round the motor cells of various cranial nerves. At the medulla nearly all the remainder of the fibres decussate with their fellows of the opposite side and then pass down the cord as the **crossed pyramidal tracts,** where they terminate at different levels by coming into relationship with the branches of the large motor cells of the anterior cornua. This communication with the cells of the cord is not a direct one, but appears to be established through short intermediary neurones in the grey substance of the cord.

Those fibres which do not decussate at the medulla travel down the anterior columns of the cord on the same side and form the **direct pyramidal tracts.** Their destination is likewise the cells of the anterior cornua, and as they descend the cord they also cross to the cells of the opposite horns. These fibres have generally all reached their destinations by the time they have arrived at the midthoracic region of the cord, below the level of which the direct pyramidal tract cannot usually be traced.

The motor paths are continued from the anterior horns outwards to the muscles by a secondary set of projection fibres known as the **lower motor neurones,** whose cell bodies are situated in the grey matter of the anterior horns. The course of these two sets of descending projection fibres is depicted in the accompanying plate (Plate 1).

In addition to the main tracts just described there are some subsidiary tracts which also conduct impulses



LATE 2.—Diagram of principal and subsidiary descending projection fibres.

downwards, and which may possibly serve in certain circumstances as additional paths for the passage of voluntary motor impulses.

The chief of these are—

1. The rubro-spinal tract (Monakow's bundle).
2. The thalamo-spinal tract.
3. The tecto-spinal tract.
4. The vestibulo-spinal tract.

1. The fibres of the **rubro-spinal tract** arise from the red nuclei which are situated in the tegmentum of the crura cerebri. They immediately decussate with their fellows of the opposite side, and then each tract passes down the lateral columns of the cord in close proximity to and just in front of the crossed pyramidal tract. The rubro-spinal tract also receives impulses from the cerebellum by fibres which pass by way of the superior cerebellar peduncle to the red nucleus.

2. The **thalamo-spinal tract** is composed of fibres which arise from the thalamus and accompany the fibres of the rubro-spinal tract downwards into the cord. Fibres from the lenticular nucleus also probably accompany this tract.

3. The **tecto-spinal tract** takes its origin from the region of the midbrain, and after decussation with the fibres from the opposite side passes down the anterior columns of the spinal cord, where it probably terminates at different levels in relation with the cells of the anterior cornua.

4. The fibres of the **vestibulo-spinal tract** arise from Deiters' nucleus, which is situated in the pons. They do not decussate, but each bundle passes down the anterior column of the cord on its own side and terminates in connection with the cells of the anterior horns. Owing to the connection which Deiters' nucleus has with the vestibular nerve through nuclei of the cerebellum, the vestibulo-spinal tract probably serves an important function in conducting impulses which assist in keeping up the tone of the muscles. It is thus seen that the cerebellum is able to exercise a direct influence on

the functions of the cord through (1) the rubro-spinal tracts by way of the superior cerebellar peduncles and (2) the vestibulo-spinal tracts by their communications with Deiters' nuclei.

CLINICAL IMPORTANCE OF THE DESCENDING OR MOTOR TRACTS

It has already been stated that nearly all the impulses which give rise to voluntary movements travel along the main descending projection tract known as the motor tract, the fibres of which pass from the cell bodies in the motor area of the cerebral cortex, through the corona radiata, internal capsule, crus cerebri, pons, and medulla (where they decussate), and thence down the crossed pyramidal tracts of the cord to reach the cells of the anterior horn. Here the fibres terminate, and the second relay, or lower motor neurone as it is called, begins and forms the rest of the path for conducting the motor impulses out to the muscles.

The tract is thus divided into two segments consisting of (1) the **upper motor neurone,** which begins in the cerebral cortex and ends in relation with the cells of the anterior horn, and (2) the **lower motor neurone,** which has its beginning in the cells of the anterior horn and its ending in the muscles. Since almost all impulses which give rise to voluntary movements pass down these tracts, it follows that any break in the course of the fibres of either of these neurones will be accompanied by some loss of voluntary power, or, to put the matter in its clinical aspect, a patient with loss of voluntary power must, except in the case of functional disorders, have a lesion somewhere in the course of the motor tracts, or in the muscles themselves, which prevents the impulses from passing.

Once, therefore, the fact of loss of power is established, the next step is to endeavour to decide whether the interruption lies in the course of the upper or of the lower neurone of the tract, and for this purpose certain diagnostic differences must be borne in mind.

The impulses which travel down the upper neurone, besides giving rise to voluntary movements, also exercise restraint over the actions of the lower neurone, so that when this restraint is removed there is found, in addition to loss of power, some rigidity of muscles and increase of the deep reflexes in the affected parts. But, inasmuch as the lower neurone is independent of the upper as far as its actual existence is concerned, there is no wasting of muscles except such as occurs from disuse, neither is there any change in their electrical reactions. On the other hand, if the lower neurone is injured there is not only loss of power but also loss of reflexes (if any happen to be situated in the affected zone), together with wasting, flaccidity, and changes in the electrical reactions of the muscles, for the nutrition of the muscles is directly dependent on the vitality of the lower segment.

These differences may be conveniently summarized in the following table :—

	LESION OF UPPER NEURONE	LESION OF LOWER NEURONE
1. *Wasting* ...	Slight, and only in consequence of disuse	Wasting a prominent feature.
2. *Reflexes* ...	Deep reflexes increased; plantar reflex gives an "extensor response"; superficial reflexes of abdomen often lost	Abolished in the affected segments.
3. *Rigidity* ..	Limbs tend to become rigid	Limbs tend to become flaccid.
4. *Electrical reactions*	No obvious changes	Reactions to faradism and galvanism modified. Frequently a typical "reaction of degeneration."
5. *Contractures*	Rigidity accompanied by contractures in which the arm tends to become flexed and adducted and the leg usually extended	Irregular deformities owing largely to unopposed actions of non-paralysed muscles.

It will be seen that increased reflexes and rigidity tend to go together, while flaccidity, wasting, and loss of reflexes generally accompany one another.

The importance of this table to the student is that in it he has the basis of all the symptoms which depend upon loss of power, and instead of endeavouring to remember a different set of points for every individual instance he can apply the general principles to his case and then vary the particulars as required.

For instance, the motor symptoms underlying primary or secondary lateral sclerosis and ataxic paraplegia will all, in so far as they depend upon a lesion of the upper neurone, obey the general rules of such lesions. Modifications will merely concern methods of onset, course, and similar details. On the other hand, poliomyelitis, progressive muscular atrophy, and peripheral neuritis have their motor symptoms founded on the principles of a lower neurone lesion.

Sometimes the two conditions are mixed, as in amyotrophic lateral sclerosis, where the usual clinical picture is that depending upon a lesion of the lower neurone as far as the hands are concerned, and upon one of the upper neurone as regards the legs, which signifies that the cells of the anterior horns in the cervical region of the cord are degenerating at the same time as those fibres of the crossed pyramidal tracts which carry voluntary impulses to the legs.

The symptoms of disease of the **subsidiary descending** tracts are less clear, but lesions which include the rubro-spinal, thalamo-spinal, and tecto-spinal fibres appear to be followed by some generalized muscular weakness, rigidity and tremors. If the lesion is above the red nucleus so that the thalamo-spinal and tecto-spinal fibres suffer while the rubro-spinal tract escapes, the principal result seems to be a tendency to develop tremors. On the other hand, when the vestibulo-spinal and other descending cerebellar tracts are affected, the principal results are loss of muscle tone and incoördination of movement. Perfection of volun-

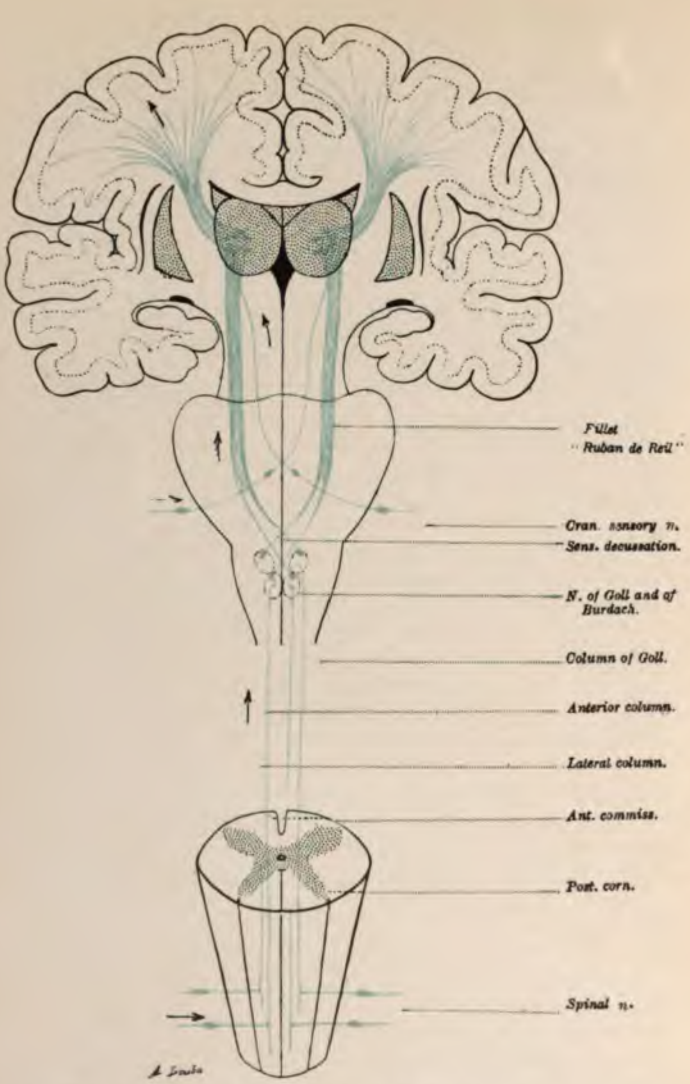


PLATE 8.—Sensory field with its principal orders of projection fibres. (*Morat.*)

tary movement depends, therefore, upon the integrity of all the descending tracts, and if any one of these is destroyed the balance is upset, with a resulting disorder of movement due partly to loss of function of the diseased tract and partly to unantagonized over-action of the tracts that remain. The symptoms of such a lesion are thus divisible into two groups—(1) *negative* symptoms due to loss of function of the diseased parts, (2) *positive* symptoms due to removal of control and consequent over-action of other systems.

For example, in a case of hemiplegia due to a lesion of the internal capsule the negative effects are shown by the inability to perform certain movements voluntarily—i.e. by "paralysis"—while the positive results are seen in the rigidity, increased tendon reflexes, and other symptoms due to unantagonized action of the cerebellar system; in some instances further positive results occur in the shape of involuntary movements (athetosis) due to unrestrained activity of the midbrain subsidiary motor systems. These instances illustrate an important principle, first enunciated by Hughlings Jackson, that a destructive lesion in any part of the nervous system tends to produce negative results due to impaired function of the part affected, and positive results due to loss of control of the injured part over other parts which are lower in the functional scale.

SENSORY NEURONES

The neurones for the conduction of sensations are more numerous and arranged in a more complicated manner than are those for movements. It is necessary to consider their arrangement and the effects produced by their injury or disease (1) in the peripheral nerves, (2) in the posterior roots, (3) in the spinal cord, and (4) in the brain.

1. SENSORY NEURONES IN THE PERIPHERAL NERVES

Head, Rivers, and Sherren, and Head and Sherren, have shown that in the peripheral system the afferent nerves can be divided into three systems—(i.) the system for deep sensibility, (ii.) the protopathic system, and (iii.) the epicritic system.

i. In the **system for deep sensibility**, sensations arising from pressure and from movements of tendons, joints, and muscles are conducted.

The locality of the stimulus and the direction of movement in a joint can be appreciated, as also can pain if the pressure is excessive or if a joint is injured. The fibres which conduct the impulses for this form of sensibility run chiefly with the nerves to the muscles, and they are not destroyed by dividing all the sensory nerves to the skin.

Thus, when the sensation to light touch is entirely lost in the skin, a sense of pressure and of movements of joints may still be appreciated provided the deep system is intact.

ii. In the **protopathic system** the fibres respond to painful cutaneous impressions and to the extremes of heat and cold.

iii. In the **epicritic system** accurate cutaneous localization of light touch, the discrimination of the points of a compass, and the recognition of the slighter differences in temperature are accomplished.

A knowledge of the existence of these three systems is obviously of the greatest importance in practical medicine.

In the first place it shows what care it is necessary to take in examining for sensibility, for when cutaneous nerves are severed an appreciation of slight pressure through the deep system may easily lead, as it often has done in the past, to an erroneous supposition that there is no anæsthesia. In testing for the tactile sensibility of the skin the very lightest form of stimulation, such as can be obtained with cotton-wool, is required.

Moreover, the loss of protopathic and of epicritic sensibility is not always equal in extent or intensity. When, for instance, union is taking place between the ends of a divided nerve the sensation to painful stimuli returns first. For a time the pain cannot be localized, but radiates widely from the spot stimulated, though always in definite directions.

Together with return of sensibility to pain there is also a return of appreciation of extremes of temperature, e.g. great heat or great cold, which Head has shown to be due to the presence of "heat" and "cold" spots. With the return of sensation for pain the nutrition of the skin also improves, and any ulcers that may have been present readily heal. Gradually the skin over the affected areas again becomes sensitive to light touch and to slight differences of temperature.

It therefore appears that the appreciation of touch and of pain is subserved by independent fibres, since Head has been able to demonstrate the dissociation of these two forms of sensation, and also that fibres which conduct the painful impressions are capable of regenerating more quickly than those which conduct the tactile impulses. Head has summarized the characteristics of the three systems as follows :*—

Loss of **epicritic sensibility** abolishes—

- Recognition of light touch over hairless parts or parts that have been shaved.
- Cutaneous localization.
- Discrimination of compass points.
- Appreciation of differences of size, including the accurate discrimination of the head from the point of a pin apart from the pain of the prick (acuæsthesia).
- Discrimination of intermediate degrees of temperature from about 25° C. to about 40° C.

Loss of **protopathic sensibility** abolishes—

- Cutaneous pain, especially that produced by pricking, burning, or freezing, together with that of stimulation with a painful interrupted current. Over hair-clad parts, plucking the hairs ceases to be painful.

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* *Brain*, Part cxvi., 1906.

Sensations of heat from temperatures higher than about 45° C.

Sensations of cold from temperatures below 20° C.

After destruction of all cutaneous afferent fibres the part is still endowed with deep sensibility.

Pressure can be recognized, and its gradual increase appreciated. Pain is produced by excessive pressure (measured by the algometer). Movements of muscles can be recognized.

The point of application of pressure can be localized.

The patient can recognize the extent and direction of movement produced passively in all the joints within the affected area.

Muscle sense.—Sensations from muscles, joints, and ligaments are generally grouped together under the term " muscle sense," and their loss, singly or combined, is largely responsible for the incoördination in locomotor ataxy.

The sense of the muscles can be tested by finding whether the patient can distinguish the differences in weight of objects of similar size; and allied to this is the power of recognizing with accuracy, by contact, the shapes of objects, i.e. the stereognostic sense.

When the sensibility of the joints is impaired the patient cannot tell in what position his limbs are placed, and this can be demonstrated by putting the limbs in various positions and requesting him (while his eyes are closed) to describe the positions or to put his other limbs into similar positions.

The sensibility of the bones can be tested by means of vibrations produced by placing on them a tuning-fork. This sensation, like those from the muscles and joints, is frequently impaired in locomotor ataxy.

2. SENSORY NEURONES IN THE POSTERIOR ROOTS

The distribution of anæsthesia in root lesions differs from that of the peripheral nerves inasmuch as the fibres are so arranged that each root corresponds to a definite segment of skin. The segmentary distribution of the posterior roots has been investigated by many observers, and with exceptional thoroughness by Head, in connection with the areas occupied by the eruption of herpes zoster, an affection which is due to lesions of the posterior root ganglia. (Figs. 2, 3.)

Destination of the fibres of the posterior roots.—The principal connections which the fibres of the posterior roots make after entering the cord may be summarized as follows :—

i. By the posterior columns of the cord with the nucleus gracilis and nucleus cuneatus of the medulla, thence by the mesial fillet to the optic thalamus, and thence to the cerebrum (this is the path for conscious muscle sense and tactile discrimination).

ii. With the cells of Clarke's column, and thence by dorsal and ventral cerebellar tracts to the cerebellum (the chief paths for maintenance of equilibrium).

iii. With the cells at the apex of the posterior horn, and thence by the spino-thalamic tract of the opposite side to the thalamus and the cerebrum (the path for pain, temperature, and diffuse tactile sensibility).

iv. With the cells at the apex of the posterior horn of each side in the same and distal segments, and thence to motor cells of the anterior horn (the path for short and long spinal reflexes).

v. With the cells of the anterior horn direct (probably the path for tendon reflexes).

3. SENSORY NEURONES IN THE SPINAL CORD

The principal sensory tracts in the cord are the posterior columns, the direct cerebellar tracts, the anterolateral tracts of Gowers, and the ascending tracts in the anterior columns.

The posterior columns.—The posterior columns are divided into posterior external (column of Burdach) and posterior internal (column of Goll). These columns are composed mainly of fibres which are a continuation of those forming the posterior roots (exogenous fibres), and to a small extent of fibres which run from one level of the cord to another (endogenous fibres).

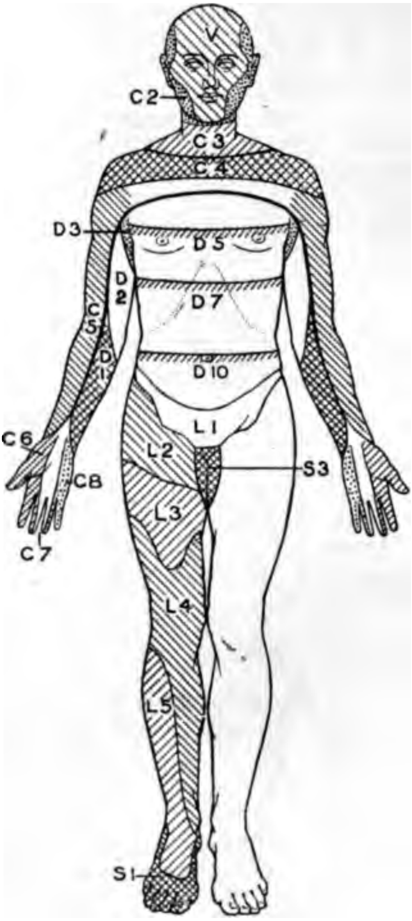

Fig. 2.—Segmentary sensory areas of the spinal cord. (*After Tooth.*)

Of the **exogenous fibres** (*a*) a large number pass directly up to the top of the same side of the cord and terminate in the nucleus gracilis and the nucleus cuneatus.

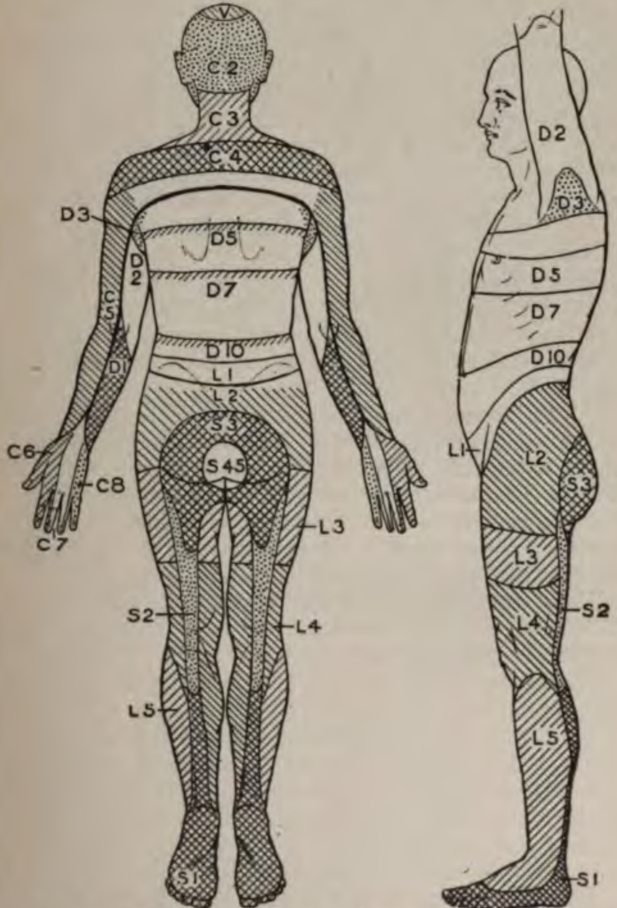

Fig. 3.—Segmentary sensory areas of the spinal cord. (*After Tooth.*)

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On their way up they give off numerous collaterals which terminate in the cells of the grey matter. From the nucleus gracilis and nucleus cuneatus there starts a second relay of fibres which cross and terminate in the optic thalamus of the opposite side. From the optic thalamus springs a third set which spreads out and terminates in the cerebral cortex. (Plate 3.)

The chief function of these long fibres of the posterior columns appears to be that of conducting impulses for tactile discrimination (i.e. compass test) and for the sensations of passive movements and position which rise into consciousness, since these sensations are not lost on the opposite side in a Brown-Séquard lesion of one half of the cord (Head).

Destruction of these columns is usually accompanied by some ataxy, though the influence in this direction of a possibly associated lesion of fibres connected with the cerebellar tracts must not be lost sight of.

(*b*) There are fibres which run a variable distance up the cord and then cross. Their function seems mainly to be that of conducting tactile impulses, and, owing to the distance they run in the cord before crossing to the ascending columns of the opposite side, they too are often apt to escape on the opposite side to the lesion in a Brown-Séquard paralysis (Head).

There are also fibres which pass up the cord some distance, and then communicate with cells at the apex of the posterior horns to form Lissauer's tract.

The fibres passing direct to the cells of the anterior horns need no special description.

The **endogenous** or **propriospinal fibres** of the posterior column which connect different segments of the cord together are situated chiefly at the concavity of the grey matter, where they form the cornuo-commissural bundle, and near the septum dividing the posterior columns of the two sides, where they form the septo-marginal bundle.

The direct cerebellar tract.—The fibres of the direct cerebellar tract take their origin from cells forming

Clarke's column which are situated on the inner side of the grey matter of the posterior horn. After passing up the cord the fibres reach the cerebellum by means of the inferior cerebellar peduncle or restiform body, and finally end in the vermis.

The origin of this tract is mainly confined to the dorsal and upper lumbar regions. Its lower limit has not been exactly defined, but in man it is probably about the first lumbar segment, and it does not increase in volume after it has reached the first dorsal segment. The fibres of this tract form an important link between those of the posterior roots and the cerebellum.

The ascending antero-lateral tracts comprise various sets of fibres; the principal are—

(*a*) The **tract of Gowers,** or **ventral cerebellar tract,** which is composed of fibres arising from cells in the grey matter and then passing upwards by way of the superior peduncle to the cerebellum, where they terminate in the vermis. The function of these fibres is similar to that of the fibres of the direct cerebellar tract, namely, to conduct impulses from the deep structures—bones, joints, ligaments, and muscles—to the cerebellum. Some fibres in this tract go also to the lateral nucleus, the corpora quadrigemina, and the optic thalamus.

(*b*) A tract of ascending fibres known as the **spino-thalamic tract,** and situated in close apposition to Gowers' tract. Its fibres also arise from cells in the grey matter, for the most part, of the opposite side of the cord, the decussation taking place in the anterior commissure. The destination of these fibres is the optic thalamus, and they form an important path for the conduction of crossed tactile impulses. Temperature and pain impulse also reach the brain by fibres of the antero-lateral tracts.

Path of impulses in the cord.—It has been shown by Henry Head and Theodore Thompson* that the grouping of the sensory impulses in the cord is quite different from that of the peripheral nerves, in which they run in three main streams by the epicritic, protopathic, and deep sensibility fibres. In the cord the impulses for the different forms of sensation tend to unite, irrespectively of the various paths by which they have arrived. Thus the tactile, painful, and thermal sensations are all collected together in their own groups, and the thermal sensations are further particularized into those for heat and cold, so that in some cases the sensations for one may be lost while those for the other are retained. This new grouping appears to take place on the same side of the cord as that by which the impulses enter, but after the new formation has taken place those of tactile sensibility, tactile localization, and painful and thermal sensibility, all cross sooner or later to the other side. But those impulses which are concerned with the knowledge of the position of the body, spatial discrimination as tested with the points of a compass, appreciation of size, form, and weight, and those conveying the sensation of vibration as produced by a tuning-fork, pass straight up the long fibres of the posterior columns of the same side to the gracilis and cuneate nuclei. They then cross at the sensory decussation and pass upwards to the optic thalamus, which part of their course is dealt with more particularly in the following section.

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* *Brain*, Part cxvi.

The main types of **cord anæsthesia** that can be recognized are those arising—

From transverse lesions.
From lesions of the grey matter.
From lesions of one half of the cord.

In lesions which extend across the cord, all forms of sensibility will be affected below the injury. If the lesion is complete, there will be total anæsthesia below its level, with probably a zone of hyperæsthesia just above, owing to irritation and inflammation of the posterior roots in the immediate vicinity of the injury.

With disease of the commissural grey matter there is very apt to be loss of sensation of pain and temperature, while that of touch is preserved—a dissociation particularly well seen in cases of syringomyelia.

When injury or disease is sharply limited to one half of the cord, as may occur in stab or bullet wounds, the symptoms known as Brown-Séquard paralysis are liable to follow: on the side of the lesion and below its level, loss of power, loss of sense of position, of tactile discrimination* and of vaso-motor tone, while on the opposite side there is loss of sensation to touch (partly), and loss of pain and temperature senses. Head has shown that while the loss of pain and temperature senses is constant in these cases, those of touch and pressure are more rarely affected—due, presumably, to their crossing to the other side of the cord less rapidly than the others.

At the level of the lesion, and on the same side, there is a zone of anæsthesia corresponding to the area supplied by those posterior roots which the lesion actually destroys, and just above the level of the lesion there is some hyperæsthesia due to irritation.

Referred pains.—In diseases of the various viscera, the pain to which they give rise is frequently "referred" to a part of the body which seemingly has no direct connection with the seat of the disease. Thus the headache from eye-strain may be situated over the forehead, and pain between the shoulders is a common accompaniment of gastric disorders.

The distribution of referred pains has been especially investigated by Head, who has constructed a scheme of intramedullary segmentation based on the hypersensitive areas which result from irritation of viscera.

The explanation is that the impulses which travel up to the cord from the diseased organs by the sympathetic fibres are reflected outwards along the sensory roots of the segment at which they happen to enter, and so the patient refers the pain to the area of distribution of these roots, and not to the viscus from which the abnormal impulses originally arise.

Ligat† has applied this principle to the diagnosis of acute abdominal conditions, and finds that by observing the hypersensitiveness of certain areas of the abdominal wall when the skin is gently pinched up, evidence can be obtained as to the particular viscus which is the seat of the disease.

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* I.e. the power to respond to the compass test.

† Hunterian Lecture, May, 1919.

4. Sensory Neurones in the Brain

A. FROM MEDULLARY NUCLEI TO OPTIC THALAMUS

In the previous sections an outline has been given of the sensory neurones of the cord and peripheral system, and of the courses which the various impulses follow. The long fibres of the posterior columns were traced up to the gracilis and cuneate nuclei in the medulla, and the various connections of the shorter ones were indicated.

At the cuneate and gracilis nuclei a second relay of neurones commences, and, after decussating with their corresponding fellows of the opposite side, the fibres pass upwards through the posterior third of the posterior limb of the internal capsule and terminate in the optic thalamus.

After their decussation they are joined in their upward course by (1) fibres conveying sensations which have already crossed lower in the cord, and (2) others conveying sensations from the head; and the combined bundle, as it ascends towards the optic thalamus, is known as the *median fillet.* It has already been mentioned that the impulses which pass up the long fibres of the posterior columns are those concerned chiefly with recognition of posture, spatial discrimination, and vibration, while those for tactile, pain, and temperature sensibility crossed earlier in their course and passed up the opposite side of the cord. It will thus be seen that, after the impulses that have passed up the posterior columns leave the medullary nuclei and cross, they again meet the impulses that have crossed lower down, so that finally all are collected together in the median fillet. The fibres, however, still maintain a definite grouping, by which in certain forms of disease one set may be affected to the exclusion of another; and the investigations of Henry Head and Gordon Holmes* show that at the medullary nuclei the impulses for the recognition of posture leave those for spatial discrimination. As far as the medullary nuclei they have travelled together up the posterior columns, but above the nuclei it is possible to find one set lost independently of the other. These observers also state that it seems that the impulses underlying the power of localizing a spot touched become detached in the brain stem from the other impulses of tactile sensation with which they have been associated in the periphery and cord.

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* *Brain*, vol. xxxiv., 1911.

B. FROM OPTIC THALAMUS TO CORTEX

At the optic thalamus another relay of sensory neurones commences, and their fibres radiate to the cortex, to which they conduct the impulses.

Present knowledge of the grouping of the different impulses as they pass from the thalamus to the cortex is largely due to the investigations of Head and Holmes, who conclude that the impulses are grouped into those representing—(1) recognition of posture and passive movement, (2) certain tactile impulses, (3) those on which the appreciation of two points depends (compass test), (4) those by which the localization of a stimulated spot is recognized, (5) temperature sensation.

C. SENSATION AND THE CEREBRAL CORTEX.

It has further been shown by Head * that the sensory activity of the cortex is not concerned with crude recognition of touch, pain, heat and cold, but that it exercises discriminative faculties as regards (*a*) recognition of spatial relations, (*b*) graduated response to stimuli of different intensity, (*c*) appreciation of similarity and difference in external objects brought into contact with the surface of the body. In cortical lesions these three aspects of sensation may be dissociated from one another, and one that is most frequently disturbed is that of knowledge of spatial relations as shown by inability to recognize passive movements of the affected part, which symptom for clinical purposes may be considered as one of the leading signs in sensory cortical disease. The localization of these cortical sensory functions is, according to Head, in the pre- and post-central convolutions, the anterior part of the superior parietal lobule, and the angular gyri. An anatomical lesion in these areas upsets the orderly sequence of physiological processes, which is shown by a disturbance of the psychical acts of sensation.

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* *Brain*, vol. xli., pt. 2, 1918.

CHAPTER III

THE REFLEXES

A **REFLEX** act consists of a contraction of a muscle or group of muscles in response to a sensory stimulus, and, as the name implies, the movement is the result of an impulse which has entered the nervous system and then been "reflected" out again. A simple reflex act is the most elementary example of a coördinated movement. In their most primitive forms reflexes always respond to the same stimulus in the same manner, i.e. their movements are stereotyped and incapable of voluntary modification; but with the development of animals of higher type the more primitive reactions become controlled by levels of higher function, so that, in many cases, some choice of reaction eventually becomes possible and the reflex movement can be modified to a varying extent according to circumstances. When the influence of the higher levels is abolished by accident or disease the reflexes below are released from this higher control and tend to return again to a more primitive state. For instance, a result of removal of one of the highest controls is seen in the "extensor response" of the plantar reflex, which is part of a more widely spread primitive movement by which the limb is withdrawn from harm. In a complete section of the cord this general flexion movement of the limb usually forms but one part of a "mass reflex" (Riddoch) which includes involuntary evacuation of bladder and rectum (*see* p. 36).

In a simple spinal reflex the impulse travels along the sensory nerves to the spinal cord, which it enters through the posterior roots, and then, passing through the grey matter of the cord, reaches the motor cells of the anterior cornua, from which impulses pass out to the muscles and give rise to movements.

The parts necessary for the production of such a reflex act are (1) a specialized end organ from which the sensory ingoing impulse originates, and which is known as the *receptor;* (2) the nerve fibres forming the paths inwards and outwards along which the impulses pass, known as the *conductor;* (3) the end organ, known as the *effector,* through which the reflected impulses accomplish their result.

The conductor, even in the simplest reflex, is composed of at least two neurones, and since the neurones, as we have already seen, are independent units, there is a junction at which the impulses are transferred from the ingoing to the outgoing neurone. This junction is termed the *synapse,* and there is some reason for believing that it is at this part of the arc that variations in the resistance to impulses take place, the direction along which they shall travel being thus determined. It is probable that no reflex act in man is really so simple as the one described above, for the collateral branches of the conductors communicate with others to form a chain far more complicated than that of two neurones.

It is obvious, from what has just been said, that for a reflex act to take place, the different parts of the arc must be intact, and it follows, therefore, that we have in testing the reflexes a valuable method of arriving at some idea of the integrity or otherwise of the parts of the arc through which the impulses travel. The behaviour of a reflex, however, does not depend solely upon the conductivity of the elements that make up the arc, for, as already stated, in normal circumstances a considerable degree of modification is exercised by impulses which arrive at the reflex arc from more distant parts of the nervous system.

On the *deep* or *tendon reflexes* this modification from a distance is mainly one of inhibition, and when, for any reason, the controlling influence is removed, the reflexes usually become exaggerated, e.g. exaggeration of the knee-jerk in disease of the pyramidal tracts of the cord.

On the *superficial reflexes* the impulses from the higher centres have somewhat different effects. In the case of the plantar reflex, removal of the higher controlling influences not only causes a more widely-spread response, but, as will be seen below, also changes the type of the reaction from one of flexion of the great toe into one of extension—the " extensor response ; " while in the case of the superficial abdominal reflexes, removal of influence from the higher centres is often associated with diminution or disappearance of the reflex, which is the opposite effect to that produced on the deep reflexes. Thus, in a case of hemiplegia, due, say, to a hæmorrhage in the region of the internal capsule, one would expect to find on the paralysed side the tendon reflexes of the arm and leg exaggerated, the superficial abdominal reflexes diminished or absent, and the plantar reflex brisk and of the extensor type.

For clinical purposes reflexes may be divided into the following classes :—

Superficial reflexes, obtained by stimulating superficial nerves of the skin.

Deep reflexes, produced by striking certain muscles or tendons.

Visceral reflexes, as those of swallowing and micturition.

Bone reflexes, obtained by percussing various bony points in the upper and lower extremity.

The **principal superficial reflexes**, with their approximate segmentation, are the following :—

Plantar reflex, movement of foot and leg produced by stimulating sole of foot. S., 1, 2, 3.

Superficial anal reflex (3rd and 4th sacral) ; this may be obtained by stimulating the skin of the perineum.

Bulbo-cavernous reflex ; this reflex, in which a contraction of the bulbous part of the urethra can be felt on stimulating the glans penis, depends on the 3rd and

4th sacral segments. The reflex is frequently lost in locomotor ataxy.

Gluteal reflex, contraction of gluteus on stimulating buttock. L., 4 and 5.

Cremasteric reflex, retraction of testicle on stimulating inner and upper aspect of thigh. L., 1 and 2.

Abdominal reflex, contraction of abdominal muscles on stimulating side of abdomen. D., 8, 9, 10, 11, 12.

Epigastric reflex, contraction of epigastrium on stimulating lower part of side of thorax. D., 4, 5, 6, 7.

Scapular reflex, contraction of scapular muscles on stimulating skin in region of scapula. C., 5, 6, 7, 8, and D., 1.

Palate reflex, contraction of soft palate when touched.

Pharyngeal reflex, contraction on stimulating posterior wall of pharynx.

Conjunctival reflex, closure of eye on touching the conjunctiva.

The ease with which these superficial reflexes can be obtained in healthy people varies considerably, especially as regards the local conditions. The plantar reflex, for instance, may be unobtainable when the sole of the foot is cold or damp, and the abdominal reflexes will not react with the same briskness in an elderly person as in a child. The degree of importance to be attached to the reactions in an individual case must, of course, be left to the judgment of the examining physician, but from general observations inferences can often be drawn concerning (1) the state of the fibres of the arc on which the particular reflex depends, and (2) the state of the upper levels of the nervous system, changes in which, as we have seen, often modify the reaction of the reflexes below.

Thus, the presence of a superficial reflex indicates the integrity of the arc through which it is produced, and so affords information which is frequently of use in localizing the level of disease in the cord.

Loss of these reflexes, on the other hand, does not necessarily mean that the arc is broken, for, in addition to this possibility, the loss may depend upon changes in the upper motor neurones, as when the abdominal reflexes are lost on the paralysed side in cases of hemiplegia, or when local conditions of the skin impair the sensitiveness of the receptors.

The **plantar reflex.**—This reflex comprises those movements of the toes, foot, and leg which take place when the sole of the foot is stimulated.

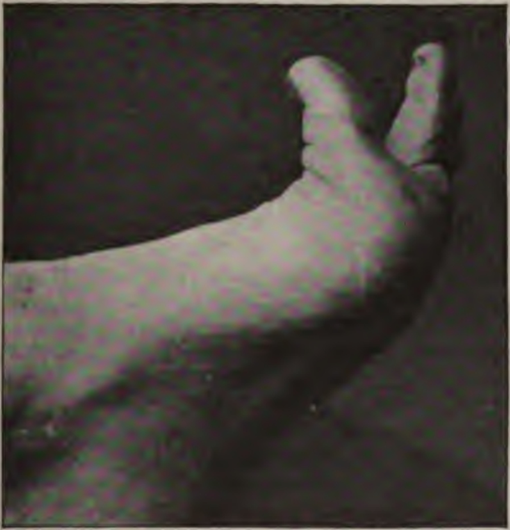

Fig. 4.—Position of the big toe in the "extensor response" (Babinski's sign).

When the sole is stimulated in the case of an infant the movement of the big toe—which for clinical purposes is the essential part of the movement to which the attention should be directed—takes place in an upward direction, i.e. the toe is extended and gives rise to what is generally known as an "extensor response," or Babinski's sign (Fig. 4). About the time the infant begins to walk this movement of extension is replaced by one of flexion; the toe now bends downwards instead of upwards, and is said to give a "flexor response." This flexor response persists during the remainder of life unless any interruption of an organic nature takes place in the conductivity of the upper motor neurone, in which case the movement returns to the original "extensor response." Therefore, from the clinical point of view, an extensor response, except in the case of an infant, indicates the presence of a lesion of the upper motor neurone.

The best method of eliciting the reflex is to draw the thumb-nail, the head of a pin, or some similar object rather firmly along the sole of the foot; in some cases the outer, and in others the inner, border of the sole gives the best results.

Oppenheim has shown that the extensor response may sometimes be obtained by stroking the inner side of the leg, and a similar reaction can also occasionally be brought about by compressing the muscles of the calf (Gordon).

To constitute a satisfactory reaction, the big toe should move slowly and decisively up or down. If, as often happens, a brisk movement of the foot and leg leads to confusion, a lighter stimulation will, perhaps, bring out the toe movement without that of the foot, or the movement of the latter may be controlled by the hand.

If the reflex is difficult to obtain or uncertain in direction, it is well to test it when the leg is loosely flexed and also when it is extended, for a reaction is sometimes better obtained in the one position and sometimes in the other. Damp or cold feet, as already mentioned, make the reflex difficult to obtain, and may lead to an erroneous conclusion that it is absent.

Value in diagnosis.—A definite "extensor response," after infancy, signifies the presence of some modification of conductivity in the pyramidal tracts, and experience shows that the obtaining of this extensor response warrants the inference that the interference is one due to organic disease, as distinguished from the functional disturbance of hysteria, neurasthenia, and allied neuroses.

Among the cases in which the extensor response is of great value are undoubtedly those of early disseminated sclerosis in which the patient complains of indefinite symptoms, numbness, tingling, with exaggerated knee-jerks and an absent abdominals, all of which would still be compatible with a diagnosis of functional disturbance.

Absence of all reflex movement whatever is suggestive of functional disease if other symptoms also point in this direction, but the same reliance cannot be placed on a negative as on a positive result, and therefore, while an extensor response indicates organic disease, neither flexor response nor absence of response necessarily excludes it.

A condition known as the "crossed plantar reflex" has been described by Byrom Bramwell and others, who noticed that in some cases of hemiplegia, in which the extensor response was present on the paralysed side, stimulation of the sole of the foot on the non-paralysed side was not only accompanied by flexor response on that side, but also by a flexor response of the toe on the opposite, i.e. the paralysed side.

The **principal deep** or **tendon reflexes**, with their approximate segmentation, are—

1. *The jaw-jerk.*—Obtained by sharply tapping the chin in a downward direction when the mouth is slightly open. The reflex is obtained through the sensory and motor divisions of the trigeminal nerve.

2. *The supinator jerk.*—Obtained by tapping the lower extremity of the radius with the forearm in a position midway between pronation and supination. The resulting movement is one of flexion of the forearm on the arm, in which the biceps, brachialis anticus, and supinator longus take part; it corresponds to the 5th cervical segment of the cord.

3. *The pronator jerk.*—Obtained by striking the lower end of the radius on its antero-internal aspect. A reflex movement of pronation results which corresponds to the level of the 6th cervical segment.

4. *The triceps jerk.*—Obtained by tapping the tendon of the triceps. A movement of extension takes place, and the level of the reflex corresponds to the 6th and 7th cervical segments.

5. *The knee-jerk* (2nd, 3rd, and 4th lumbar segments). —Full particulars of this reflex are given below.

6. *The tendo-Achillis jerk.*—Obtained by tapping the Achillis tendon. The patient should kneel in a chair with the feet over the edge, in which position a smart tap on the tendon is followed by a jerk of the foot. It corresponds to the 1st sacral segment.

These reflexes are generally exaggerated when the pyramidal tracts above their level are diseased, while they are diminished or lost when the normal inhibitory influence of the pyramidal tracts is increased or when the continuity of the reflex arc is interrupted. Their reaction in different circumstances may be exemplified by a detailed consideration of the knee-jerk.

The knee-jerk.—This reflex is obtained by sharply striking the tendon of the quadriceps extensor just below the patella, care being taken that all the surrounding muscles are relaxed.

In doubtful cases relaxation is best ensured if the patient clasps his hands together and pulls one against the other, his eyes being meanwhile raised towards the ceiling. This action, known as Jendrassik's method of reinforcement, occupies the attention and tends to remove any involuntary stiffening of the muscles.

Absence of the knee-jerk indicates organic disease, as also does inequality, but an increase may be found in functional as well as in organic disturbances.

The reflex arc for the knee-jerk is situated at the level of the 2nd, 3rd, and 4th lumbar roots, and anything which impairs the conductivity of the elements of this arc tends to diminish the reflex. Thus in peripheral neuritis, where the mixed nerve is at fault, the afferent impulses are blocked by the inflamed or degenerated peripheral sensory nerves, and even if they reach the cord the chances are that the motor fibres are unable to conduct them outwards. In locomotor ataxy the impulses are arrested at the posterior roots, while in poliomyelitis (of the lumbar region) the break occurs at the cells of the anterior horns. Impulses passing down the pyramidal tracts are constantly inhibiting the knee-jerks, so that to make the mechanism complete it is necessary to consider these fibres in connection with those of the lower arc.*

An interruption in the continuity of the pyramidal fibres is accompanied by removal of the normal inhibition, and thus the knee-jerk is exaggerated, e.g. in lateral sclerosis, transverse myelitis (above the lumbar region), and other diseases in which the pyramidal tracts are affected. On the other hand, excitation of the pyramidal fibres tends to increase their inhibitory influence, and diminishes the knee-jerk, as may be seen in some cases of meningitis and cerebral tumours.

To sum up: The **knee-jerks are lost** (Fig. 5)—

A. From *interruption of the reflex arc.*

1. Injuries or disease of the lumbar cord causing destruction of the "centre."
2. Diseases interrupting impulses to or from the lumbar region of the cord, e.g. affections of anterior or posterior roots, mixed nerves, or muscles.

B. From *inhibition due to irritation of the pyramidal fibres*, e.g. during early stages of cerebral hæmorrhage and fracture-dislocation of the spine, and in some stages of meningitis. In many instances, e.g. cerebral hæmorrhage, the inhibition is only temporary, and the lost or diminished knee-jerk is soon succeeded by one that is exaggerated.

The **knee-jerks are increased** (Fig. 5)—

A. From *interruption in the conductivity of the pyramidal fibres above the lumbar region*, whereby the normal

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* Sometimes it happens that the peripheral ingoing and outgoing fibres are extraordinarily irritable for a short time before their conductivity is lost, and so the knee-jerks are occasionally found to be increased for a brief period during early stages of locomotor ataxy and peripheral neuritis. Such cases are, however, quite exceptional.

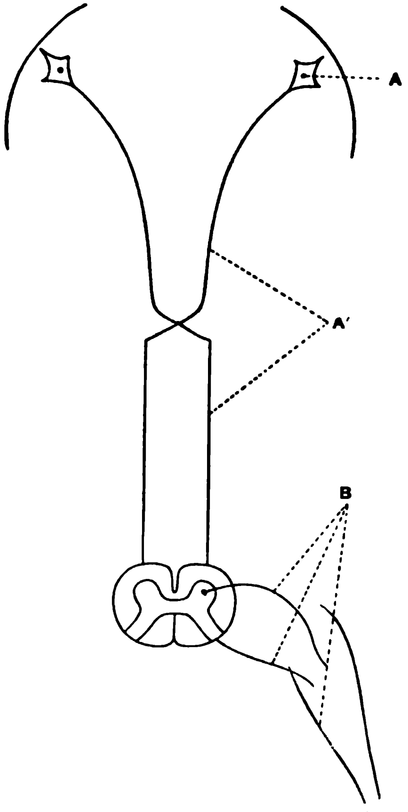

Fig. 5.—Diagram to show causes of main variations in the knee-jerk.

In lesions of the upper neurone (A and A′) the knee-jerk is usually increased
In lesions of the lower neurones or of the quadriceps extensor muscles (B) the knee-jerk is usually lost.

inhibition is removed. This may be due to organic disease, e.g.:

1. Traumatic, as in fracture-dislocation of the spine.

2. Inflammatory, as in transverse myelitis.
3. Degenerative, as in spastic paraplegia.
4. The lesion may be in the brain, as in cases of hemiplegia.

B. In *functional disease.* In many cases of neurasthenia and hysteria the knee-jerk is increased.

It will be observed that an exaggeration of the knee-jerk may accompany both functional and organic disease, but that absence of the knee-jerk always signifies the existence of an organic lesion.

The knee-jerks in cases of complete division of the cord.—In cases where the continuity of the spinal cord is *completely divided*, by injury or disease, so that there is a total interruption of all impulses at the level of the lesion, the knee-jerks, instead of being exaggerated as they are when the pyramidal tracts alone are involved in partial transverse lesions, are lost. Formerly it was considered that this loss was always permanent, but careful observation of series of cases in which the cord had been completely divided by injuries during the War has necessitated some modification of this view, for Riddoch* has shown that in certain uncomplicated cases of complete division of the cord, after the initial shock has passed off, recovery of irritability of the isolated portion with return of knee-jerks, though of poor quality, may take place, provided the general health of the patient remains good. If, however, the tone of the nervous system is lowered by ill-health, as by septicæmic infection which so often arises in these cases from cystitis, pyelitis, and bedsores, then the knee-jerks either do not return or, having returned, die away again as the septicæmia develops.

The **principal visceral reflexes** to be considered are those of the pharynx, bladder, and rectum.

Pharyngeal and laryngeal reflexes.—When these reflexes are impaired there is difficulty in swallowing and coughing. The food is apt to pass into the larynx without exciting cough, and the patient may be choked, or may suffer from broncho-pneumonia following irritation of the lungs.

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* *Brain*, vol. xi., 1918.

Bladder and rectal reflexes.—When the continuity of the spinal cord is interrupted above the lumbar enlargement the reflex arc for the bladder is cut off from all communication with the brain. Sensory impulses from the full bladder can no longer pass upwards, nor can impulses pass down to control the sphincter. The bladder, therefore, has to depend upon its own reflex, and it either fills and discharges automatically, or, as frequently happens, the discharging reflex movement is not completely carried out, and the bladder becomes over-distended and finally overflows, producing "false" incontinence. When the disease has destroyed the lumbar enlargement of the cord the reflex is lost; the walls of the bladder then lose their tone and cannot retain the urine, which continually runs away. Incontinence may also occur from lesions of the cauda equina.

The usual result of a cord lesion upon the rectal sphincter is constipation and involuntary passage of fæces.

The mass reflex.—The phenomenon known as the "mass reflex," first described by Head and Riddoch,* is a combined reflex action of the lower limbs, abdominal wall, bladder, and sweat-glands, which occurs in certain cases of severe injury to the cord.

Uncomplicated cases of gross injury to the spinal cord are followed by a period of shock varying in duration from a few days to three weeks, during which time there is complete loss of function of the parts below the lesion. The limbs lie motionless and flaccid, and sensation is lost. Reflex action in the isolated section of the cord is almost entirely abolished, and associated with this loss are retention of urine and fæces and dryness of the skin. As the effects of shock pass off, this first stage, known as the "stage of flaccidity," is succeeded by a "stage of reflex activity," in which the paralysed muscles recover their tone, the bladder and rectum commence to act automatically, and an area of skin varying with the level of the lesion may begin to sweat. During this stage of reflex activity, in a well-defined group of cases, especially when the spinal cord has been completely divided, it has been found that a nocuous stimulus, such as a scratch or a prick of any part of the paralysed limbs, is followed by a flexor spasm of the legs at the hip, knee, and ankle, with extension of the toes, and also, if the lesion is high enough, by a flexor spasm of the trunk from contraction of the muscles of the abdominal wall. When the stimulus is fairly strong or applied to some spot in the midline of the body, e.g. the region of the perineum, the flexor spasm is often bilateral, there being a simultaneous contraction of both legs and of the abdominal wall. Moreover, the effects of the stimulus may spread to other parts of the cord and set the bladder and skin reflexes in action, thus producing evacuation of urine and sweating over a skin area corresponding to the level of the lesion. It is to this combined reflex action that the term *mass reflex* is given. When the bladder has begun to discharge automatically but is still unable to evacute its contents completely, Riddoch* has shown that the "mass reflex" may sometimes be utilized to complete the evacuation and so obviate the necessity of catheterization for the removal of the residual urine. The interval at which the bladder tends to discharge itself automatically must be noted for each patient, and at about the time it is due to evacuate its contents the "mass reflex" is produced by stimulating the abdominal wall or the lower limbs, with the result that the reflex activity of the bladder is reinforced and its contents entirely emptied into the bottle. Thus, apart from the avoidance of catheterization, the risk of wet beds is much diminished, and the patient is often able to carry out the whole process by himself.

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* *Brain*, 1917.

Rhythmical clonic contractions of certain muscles can often be obtained when there is a lesion of the upper neurone.

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* *Lancet*, Dec. 21, 1918.

Ankle-clonus is obtained by suddenly flexing the foot at the ankle-joint, when, on keeping up the pressure, the foot gives a series of jerks against the hand owing to the clonic contraction of the calf muscles. An ankle-clonus does not exist in health, but is often obtained when the pyramidal tracts are degenerated; hence it is frequently associated with an increased knee-jerk and an extensor plantar response.

Sometimes, on trying to obtain an ankle-clonus, a few irregular contractions occur as the foot is being flexed, but cease when the pressure is increased and maintained. These are known as "spurious" ankle-clonus and do not in themselves indicate an organic lesion, although they do not, of course, negative the possible existence of one. The importance of an abortive clonus of this kind must be judged in every individual case according to the other symptoms that may be present.

When the degeneration of the motor tracts is extensive, clonus can often be obtained in other situations, such as at the wrist, elbow, and jaw. When the knee-jerk is greatly exaggerated, a patellar clonus can often be elicited by suddenly pushing the patella in a downward direction, and so starting clonic contractions in the quadriceps extensor.

CHAPTER IV

RIGIDITY AND CONTRACTURES

When the conductivity of the upper motor neurones is impaired by injury or disease, e.g. in cases of hemiplegia or myelitis, there ensues a rigidity of certain groups of muscles which, if unchecked, is apt to be followed by permanent contractures of the affected limbs.

Thus, in the later stages of hemiplegia the arm is carried in a characteristic attitude across the chest, being drawn close to the side by adduction and flexed at the elbow, wrist, and fingers, while the leg is kept stiff and moved as one piece from the hip. In the cerebral diplegia of children there may be sufficient spasm of the adductors of the thigh to cause the legs to cross one another, and so to produce the "scissors-legged" form of progression when an attempt is made to walk. (Fig. 86, p. 331.) In some cases of paraplegia from severe transverse lesions of the cord the legs become flexed instead of extended.

In the later stages of rigidity, degenerative changes, followed by contraction, take place in the fibres of the muscles themselves and contribute further to permanent fixation of the joints.

The cause of rigidity, which usually comes on slowly after lesions of the upper motor neurones, is somewhat obscure, but, broadly speaking, it may be ascribed to the unantagonized influence of impulses from the subsidiary descending tracts, and especially from the cerebellum after impulses from the cerebrum have been lost. This view was many years ago presented by Hughlings

Jackson, who propounded the hypothesis that the cerebrum is the centre for changing movements and the cerebellum for continuous tonic movements, and that when the impulses from the cerebrum are cut off, those from the cerebellum come into undue predominance. The subject has lately been more particularly studied from the numerous cases of paraplegia due to injury of the spinal cord which have been observed during the War, and it has been shown that when the lesion is mainly limited to the crossed pyramidal tracts the resulting position of the lower limbs resembles that of an ordinary hemiplegia. This condition is known as *paraplegia in extension*. If, however, in addition to the interference with the continuity of the main pyramidal tracts, the lesion is sufficiently severe also to destroy the conductivity of the subsidiary motor tracts (rubro-spinal and vestibulo-spinal), the tone of the extensor muscles completely disappears and, on stimulating the limb, the only reaction that can be elicited is a movement of flexion. This state is known as *paraplegia in flexion*. If allowed to progress unchecked by treatment it may finally end in permanent contractions of the limbs in the flexed position. From the clinical point of view it will thus be seen that a " paraplegia in flexion," with which is frequently associated the " mass reflex " (*see* p. 36), is an indication of very severe damage, even if not a complete division, of the structures of the spinal cord.

Experimentally the subject comes under the heading of **decerebrate rigidity**, which Sherrington defines as a " condition which ensues on removal of the forebrain by transection at any of the various levels in the mesencephalon or the thalamencephalon in its hinder parts."

The muscles mainly affected in decerebrate rigidity are those which, when the animal is in the vertical position, tend to counteract the force of gravity; and Sherrington points out that two separate systems of motor innervation can thus be recognized, one exhibiting "those transient phases of heightened reaction which constitute reflex movements," while "the other maintains that steady tonic response which supplies the muscular tension necessary to attitude." Sherrington considers that Hughlings Jackson's view of the causation of these rigidities in man is in its general sense supported by the results of more recent investigation, for, though the forces which Hughlings Jackson attributed to the cerebellum may eventually be traced to the afferent nerves of muscles and to the nerve of the otic labyrinth, the existence and general relationship of the "clonic" or "phasic" (Sherrington) and tonic systems remain the same.

Prevention of rigidity.—In all cases it is most important to try to prevent the onset of rigidity and contracture, the latter of which, when once established, will effectually prevent any return of power that a partial recovery of the functions of the injured nerve fibres might otherwise allow.

Massage, electricity, and passive movements are the methods upon which reliance must be placed, and if these are to be of real use they must be practised with regularity and perseverance.

Massage maintains the vitality of the muscles and keeps them supple, the passive movements prevent the formation of adhesions round the joints, and electricity, judiciously applied, affords a further aid to both. Care must be taken to correct the tendency which a limb, e.g. an arm, may show to remain in positions of flexion and adduction during the intervals of treatment, and, if necessary, it should be fixed in a favourable position, especially during the night, by suitable splints or supports.

The masseur should pay particular attention to the nutrition and development of the weaker groups of muscles, e.g. the abductors and extensors of the arm, so as to aid these as far as possible in resisting the greater power of the flexors and adductors.

Persistent treatment on these principles will do much towards averting the onset of rigidity, and will often make an immense amount of difference to the ultimate utility of a limb.

In lower neurone lesions also, e.g. in the case of infantile paralysis, deformities are apt to arise from the fibrosis and subsequent contractions of the paralysed muscles, as well as from the unantagonized action of the healthy muscles.

Here, again, a great deal of trouble may be obviated by massage, passive movements, and electrical treatment, care being taken to keep the limb in suitable positions during the intervals of active treatment. The contractions in cases of this class can often be improved by some operation by which the tendons are cut or lengthened, but in most instances it is necessary also that the massage should be thoroughly carried out afterwards, otherwise but temporary benefit may be obtained from the operation.

Structure and function of muscle substance.—Muscle fibres are composed of two contractile substances, one fibrillar, which is easily excited, and the other sarcoplasmic, of low contractility. Rapidity of movement has been shown to depend upon the degree of differentiation of the fibrillar elements, which are considered to be a higher form developmentally than the sarcoplasmic substance (Ramsay Hunt), and the reaction of muscles in various circumstances and their behaviour in many diseases are probably influenced by changes in the proportionate amount and quality of these substances. For instance, it has been suggested that the reaction of degeneration is the result of disappearance of fibrillar substance, accompanied by an undue increase of sarcoplasmic substance, so that the reaction is that of sarcoplasm; and the hypothesis has also been brought forward that the weakness of muscles in certain " functional " disorders may be due to a lack of proportionate contraction of the two substances.

CHAPTER V

ELECTRICAL REACTIONS

NORMALLY, contractions of a muscle can be obtained by stimulating its nerve or the muscle fibres themselves by the faradic (interrupted) or galvanic (constant) current.

If, with the interrupted current, one pole is put on some indifferent part of the body, and the other on the muscle to be tested, sharp contractions occur in response to the stimuli, and, if the latter succeed one another with sufficient rapidity, the muscle is tetanized. The maximum effect is obtained when the electrode is placed upon certain spots known as motor points. It is a matter of indifference which pole is used.

Contractions from the galvanic (constant) current can also be obtained through the nerves or muscle fibres, but only when the contact is made or broken.

In this form of stimulation there is choice of pole to be observed, for contraction is most easily obtained on making contact between the muscle and the negative (kathode) pole, as expressed by the statement that KCC is normally greater than ACC.

When the communications between the muscle and its corresponding motor cells in the cord are broken, as they are in many lower neurone lesions, certain distinctive modifications, known as the **reaction of degeneration,** are apt to occur.

The main features of this change are:

(1) *As to the nerve.*—Loss of excitability to both faradic and galvanic current, so that stimulation ceases to produce any result.

(2) *As to the muscle.*—Loss of contraction to faradic stimulation while to galvanism the muscle, though it may for a time be unduly irritable, contracts slowly and in an abnormal manner to the two poles. If no recovery takes place the contractility gradually diminishes, and eventually, when the degeneration of the muscle fibres is complete, no reaction of any kind can be obtained. The reaction of degeneration also usually includes polar changes, whereby a contraction is more easily obtained by establishing contact with the positive (anode) than the negative (kathode) pole (usually described as ACC being greater than KCC), but experience has shown that these changes are not so important for diagnostic purposes as they were formerly thought to be.

When the fibres of a muscle do not waste uniformly, the reaction at any given time will be proportionate to the number of healthy fibres remaining, and thus there will probably be a diminished reaction to both currents, with no such qualitative changes as usually accompany the reaction of degeneration.*

The use of condenser discharges for the testing of nerves and muscles has more recently been introduced. The advantages claimed for this method of testing are that it is more rapid, more accurate, and that more information can be obtained from its use.

Therapeutic uses of electricity.—In diseases of the nervous system electricity is chiefly used (1) as a general tonic, (2) to keep up the vitality of wasting muscles, (3) to relieve pain.

As a general tonic it is used as an adjunct to other forms of treatment in hysteria, neurasthenia, and nervous debility of any kind, and the choice lies chiefly between the interrupted, constant, high-frequency, and static forms.

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* Other less common variations in the electrical reactions are alluded to under the headings of the diseases in which they occur.

The interrupted and the high-frequency currents are best when general stimulation is required. They appear to produce a certain amount of improvement in nutrition, partly, no doubt, by increasing the activity of the vaso-motor apparatus. They also act locally as a counter-irritant.

For the relief of pain the constant current is mostly employed. The positive pole (anode) should be applied to the painful part, since it has been shown to produce a greater sedative effect than the negative pole (kathode). When the two poles are kept stationary (the so-called *stabile* application) the current is less stimulating than when the electrode is moved (the so-called *labile* application); this fact therefore enables a further modification to be made in treating painful states. The pains of neuritis, sciatica, and various other forms of peripheral nerve lesions are often relieved by electrical treatment, and neuralgias and headaches are occasionally dispersed by the application of high-frequency currents.

In treating disorders of sensation, e.g. the anæsthesias of hysteria, the application may often best be made by a wire brush.

The maintenance of the nutrition of wasting muscles is one of the most valuable functions of electricity. For this purpose the interrupted current is generally the most useful. An idea has been prevalent that the interrupted current should be applied only when the muscle contracts to it, and that in other cases preference should be given to the constant form, but it was shown by Lewis Jones that there is no adequate reason for holding strictly to this view. Moreover, cases showing the reaction of degeneration seem to have progressed quite as well with the one as with the other.

This question of the choice of current is important, since it is usually far simpler for the patient to use and maintain a faradic battery.

The application of static electricity is credited with producing improvement in general metabolism.

CHAPTER VI

THE AUTONOMIC SYSTEM (SYMPATHETIC AND PARASYMPATHETIC SYSTEMS)

The Autonomic (or Vegetative) System forms the nervous mechanism by which the chief functions of life are performed outside the conscious mind.

The autonomic system consists of two divisions—(1) the *vertebral sympathetic,* which comprises the chain of ganglia situated alongside the vertebral column; and (2) the *parasympathetic* (autonomic system of Langley), which takes its origin from three principal areas, namely, the midbrain, the medulla, and the sacral region. The efferent fibres of these two systems are mainly distributed to the unstriped muscle fibres of the heart and vessels, to glands, e.g. the liver and pancreas, and to the skin and viscera, e.g. the stomach, intestine, and bladder. Most of these structures receive a double nerve supply—one from the vertebral sympathetic and one from the parasympathetic system—and the effects of physiological and pharmacological experiments tend to show that the actions of these two systems are mutually antagonistic. Thus, the pupil is contracted through the parasympathetic fibres and dilated through the cervical sympathetic; the heart is inhibited through the parasympathetic fibres of the vagus and accelerated through the accelerator fibres of the vertebral sympathetic; the constrictors of the blood-vessels are contained in the vertebral sympathetic, and the dilators (where they exist) principally in the parasympathetic system.

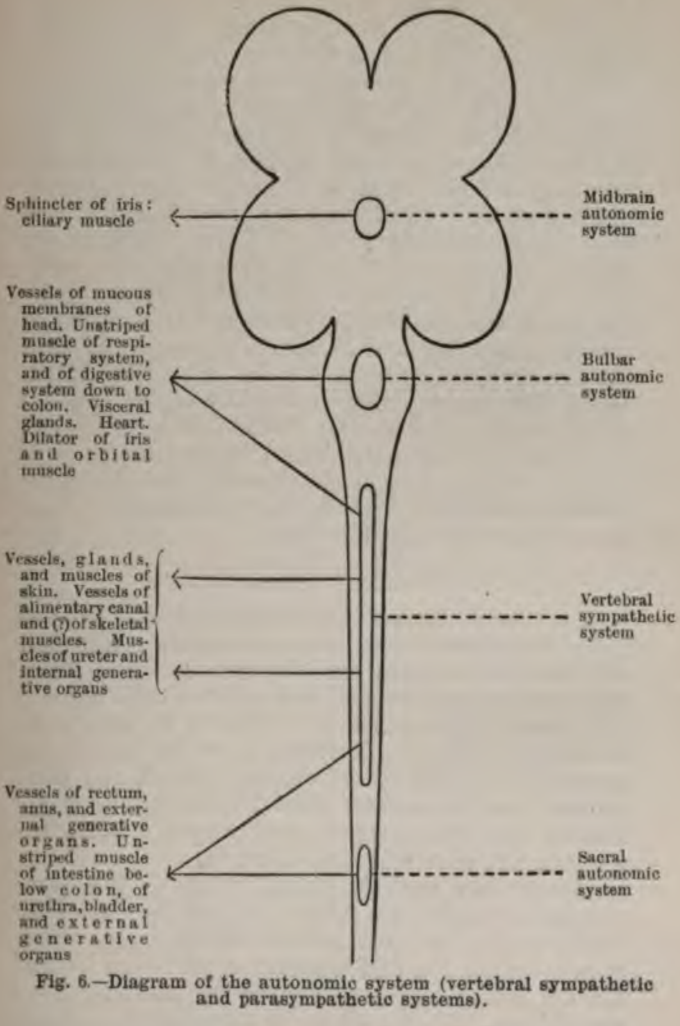
Sphincter of iris: ciliary muscle
Midbrain autonomic system
Vessels of mucous membranes of head. Unstriped muscle of respiratory system, and of digestive system down to colon. Visceral glands. Heart. Dilator of iris and orbital muscle
Bulbar autonomic system
Vessels, glands, and muscles of skin. Vessels of alimentary canal and (?) of skeletal muscles. Muscles of ureter and internal generative organs
Vertebral sympathetic system
Vessels of rectum, anus, and external generative organs. Unstriped muscle of intestine below colon, of urethra, bladder, and external generative organs
Sacral autonomic system

Fig. 6.—Diagram of the autonomic system (vertebral sympathetic and parasympathetic systems).

Broadly speaking, the function of the parasympathetic system is to store up energy (anabolic), while that of the vertebral sympathetic is to set it free (katabolic). States of mind such as fear, pain, and anger, for instance, at once demand energy for purposes of bodily defence, which energy is rapidly set in motion and results in such activities as dilatation of the pupils, trembling of limbs, rapid beating of the heart, and rise of blood-pressure, all of which are associated with ideas of self-preservation by attack or flight. The functions of the autonomic system (sympathetic and parasympathetic) are also intimately related to those of the ductless glands (endocrinic system), the secretions of which regulate the balance normally existing between the two divisions (sympathetic and parasympathetic). It will thus be seen how this balance may be upset by an excessive call on one set of endocrinic glands, e.g. the thyroid or suprarenal, as may occur through great fright or horror, or from some primary disorder of the glands themselves by which their compensatory functions become diminished or lost.

THE PARASYMPATHETIC SYSTEM

The efferent fibres of this system are medullated, and those arising from the midbrain and medulla run to their destination in company with the cranial nerves. Thus, the constrictors of the pupil run with the third nerve to the ciliary ganglion and then pass on to supply the sphincter of the iris. Secretory fibres for the salivary glands, in the form of the chorda tympani, and other secretory and vaso-dilator fibres for the regions of the mouth, nose, and pharynx accompany the facial, trigeminal, and glosso-pharyngeal nerves.

Among the most important fibres of the parasympathetic system are those which pass by way of the vagus to the unstriped muscles of the heart, blood-vessels, œsophagus, and intestines down to the level of the descending colon, and also to the pancreas, liver, and kidneys. The remainder of the colon, the rectum,

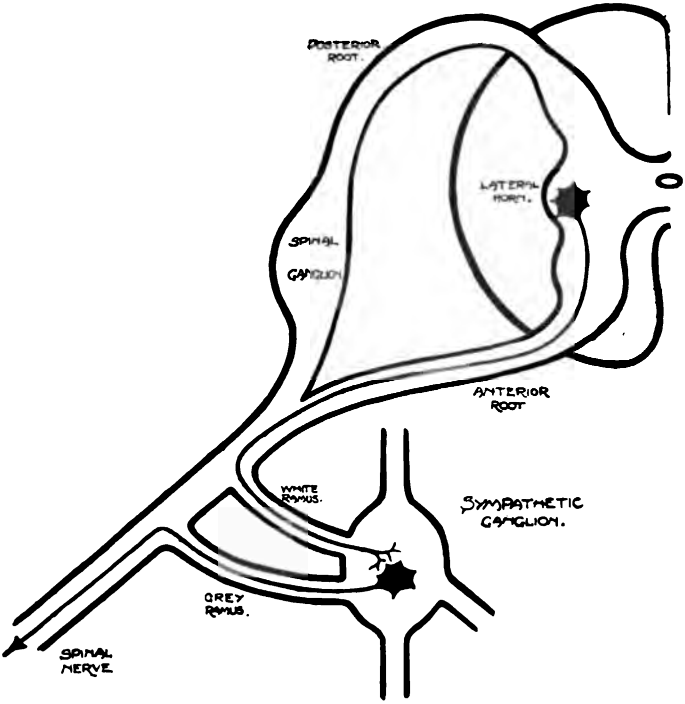

PLATE 4.—Diagram of arrangement of vertebral sympathetic system.

bladder, and genital organs receive their supply of parasympathetic fibres from the sacral source which passes out from the cord by the 1st sacral root.

The Vertebral Sympathetic System

The efferent fibres of the vertebral sympathetic system arise from groups of cells known as the intermedio-lateral tract, which are situated in the concavity of the ventral horns of the spinal cord, whence the fibres pass, as the white rami-communicantes, into the ganglion cells at the side of the vertebræ. From these ganglion cells emerge fibres known as the grey rami-communicantes, which join the fibres of the anterior roots, and thence pass down the mixed nerves to their various destinations. (Plate 4.) The fibres forming the chain of the cervical sympathetic arise from the 1st and 2nd dorsal segments of the cord.

The vertebral sympathetic system is constantly stimulated by adrenalin, which is a product of pigmented cells known as chromaffin or chromaphil cells, and is secreted by the medullary portion of the suprarenal body. It is probable that the parasympathetic system is stimulated by some substance in a similar manner. In this way a certain " tone " of the two systems is established, and if for any reason the balance between the two is upset, either by heightened tone of one or lowered tone of the other, symptoms of disease are produced. An excessive tone of the parasympathetic system is spoken of as vagotonus, while a similar condition of the sympathetic is known as sympatheticotonus.

The symptoms of disturbance of the sympathetic and parasympathetic systems are very numerous, and include many of those of a so-called functional nature, as, for example, flushing, tingling, sensations of giddiness, and "sinking" feelings. Vaso-motor symptoms are particularly common, and may arise from disturbances in the brain, medulla, or spinal cord. For example, emotional disturbances in the brain may cause pallor or blushing; vaso-motor disturbances of the medulla give rise to

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various symptoms, one of which is a disorder of the heart known as a vaso-vagal (Gowers) or pseudo-anginal attack, characterized by palpitation, tachycardia, and a sense of suffocation. In lesions of the spinal cord, anterior roots, or mixed nerves there may be vaso-motor symptoms, as, for example, is seen in some cases of peripheral neuritis and poliomyelitis.

The different reactions of the sympathetic and parasympathetic systems have been demonstrated pharmacologically by various drugs. Adrenalin, for instance, as already mentioned, stimulates the vertebral sympathetic and causes the blood-pressure to rise, the pupils to dilate, and the muscles of the hairs to contract. It also stimulates the salivary glands and excites various areas of unstriped muscle tissue.

Clinically, disturbances of these systems may often be inferred from signs of vaso-motor instability, as shown in the condition known as dermographism, in which a blush is produced out of proportion, both in its intensity and duration, to the stimulus which caused it. Such instability is well seen in people subject to urticaria, and a somewhat similar condition, known as the *tache cérébrale*, is frequently met with in cases of meningitis.

In other instances stimulation of the skin is followed by pallor instead of redness.

The adaptability of vaso-motor reactions may also be tested by taking the blood-pressure and pulse-rate when the patient is in different positions, such as lying down and standing up.

The functions of the cardiac parasympathetic system may be tested by observing the condition known as respiratory arrhythmia of the pulse and by testing the oculo-cardiac reflex. Normally the heart beats more rapidly and the pulse becomes smaller during inspiration, while the reverse takes place with expiration; but if this arrhythmia becomes very noticeable or takes place with slight respiratory movements it may be considered abnormal, and is probably to be looked upon as indicating undue irritability of the medullary centres. The oculo-cardiac reflex is the slowing of the heart that follows compression of the eyeballs. It is said to occur in from 60 to 80 per cent. of healthy people to the extent of slowing the heart six or eight beats in the minute. Excessive slowing appears to indicate some functional derangement of the nervous control exercised by the vagus over the heart, which is suggestive of a condition of vagotonus.

THE CERVICAL SYMPATHETIC TRUNK

Anatomical.—The fibres of the vertebral sympathetic system for the head and neck emerge from the upper dorsal segments of the cord. Situated on the trunk as it passes up the neck are two and sometimes three ganglia from which nerve fibres pass to their various destinations.

The cervical sympathetic cord supplies (1) *motor* fibres to the dilator muscle of the iris, to the unstriated fibres in the upper eyelids, and to Müller's muscle, which consists of a small collection of unstriped fibres that pass across the spheno-maxillary fissure at the back of the orbit; (2) *secretory* fibres to the sweat-glands of the head and neck and to the submaxillary salivary glands; (3) *vaso-motor* fibres to the vessels of the head and neck and arms; (4) *pilo-motor* fibres to the skin of the head and neck; (5) *accelerator* fibres to the heart.

The fibres which cause dilatation of the pupils arise in the medulla and pass down the spinal cord to the cilio-spinal centre, which is situated in the cervical region. They may thus be injured before they leave the cord. They emerge from the cord in company with the anterior roots of the upper dorsal nerves, and, after passing up the cervical sympathetic to the Gasserian ganglion, reach the pupil by the long ciliary branches of the 5th nerve. Their action may be tested by pinching or otherwise stimulating the back of the neck, as a result of which dilatation of the pupils should occur.

The vaso-motor fibres for the head and neck appear consequence of tuberculosis.

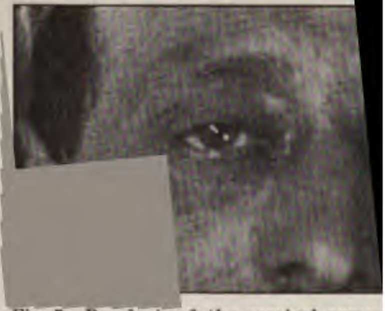

Fig. 7.—Paralysis of the cervical sympa
side. Note the ptosis and enophthalm
also the pupil is smaller.

Symptoms. *Ptosis.*—The palpebral
from paralysis of the unstriped m
lid, which causes the lid to droop (Fi

Enophthalmos.—Paralysis of Mül
lowed by some gradual retraction of

Pupil.—The pupil on the affecte
from the unantagonized action of the
which is supplied from the autonor
3rd nerve and ciliary ganglion. I
when it is shaded or when the cerv
stimulated in the neck

Anhidrosis.—Absence of sweating on the side of the paralysis may be present, but it is not a constant symptom, and hyperhidrosis sometimes occurs.

If the cervical sympathetic is irritated the signs are reversed, but symptoms of irritation are less frequently met with than those of paralysis.

Treatment of affections of the autonomic system.—The treatment of these affections is not very satisfactory. The tone of the tissues supplied by the vertebral sympathetic system is normally kept up by a secretion from the medulla of the suprarenal body, so that adrenalin may be given in certain cases where there is reason to think a lowering of tone has taken place.

The secretions of other ductless glands besides the suprarenal body exercise an influence over the functions of the vertebral sympathetic, and it is therefore well to bear in mind that disorder of these glands may sometimes be the cause of sympathetic disturbance, and that in such cases a judicious administration of preparations of, for example, the thyroid or pituitary body may sometimes give relief. Vaso-motor symptoms which accompany such diseases as poliomyelitis and peripheral neuritis may sometimes be to some extent relieved by local treatment, e.g. massage and electricity.

CHAPTER VII

GENERAL FUNCTIONS OF THE BRAIN

I. THE CEREBRUM

THE principal functions of the cerebrum are to coördinate impulses received from the external world and to originate action accordingly. The cerebrum thus acts as the organ of discrimination through which the individual is enabled to adjust himself to changing circumstances. It is especially concerned with the discrimination of those impulses which exercise their influence from a distance through the mechanism for vision, hearing, and smell, and on this account the cerebrum has been described by Sherrington as "the ganglion of the distance receptors."

The general sensory impulses are conveyed to the cerebrum and the motor impulses are conveyed away from it by long bundles of fibres known as projection systems, a description of which has already been given (p. 5 *et seq.*). These different projection systems are linked up with one another and with other parts of the brain by association fibres (Fig. 8), and impulses pass along these to reach the higher centres, where they form the basis of the psychic processes.

Bolton divides the functions of the cerebrum into three grades, as follows :—

1. *Projection spheres*, or regions to which afferent sensorial impressions pass from the parts of the body devoted to the appreciation of the types of specialized sensation, namely, vision, hearing, bodily sensibility, smell, and taste:

2. *Centres of lower association*, which lie in the immediate neighbourhood of each of the areas included in the first group, and which subserve the elaboration of the different varieties of sensorial impressions into simple perceptions and the association of these psychic units into higher complexes.

3. *The centre of higher association and coördination* in the prefrontal region, which subserves the grouping of these higher complexes into harmonious series of concepts by means of voluntary attention and selection.

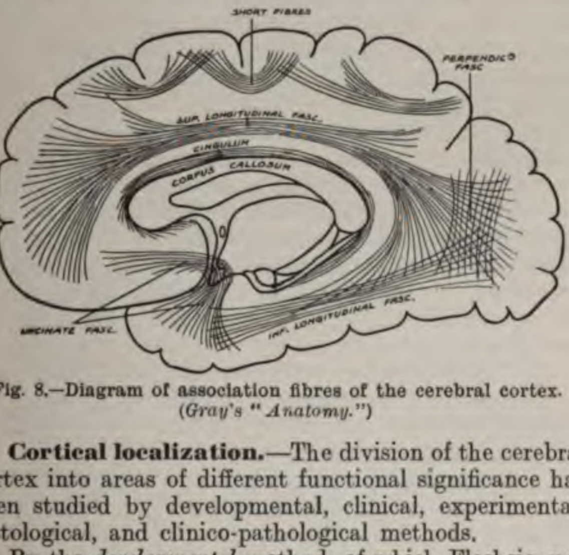

Fig. 8.—Diagram of association fibres of the cerebral cortex. (*Gray's "Anatomy."*)

Cortical localization.—The division of the cerebral cortex into areas of different functional significance has been studied by developmental, clinical, experimental, histological, and clinico-pathological methods.

By the *developmental* method, of which Flechsig was the principal exponent, the brain can be mapped out into areas corresponding to the different periods at which myelinization of the fibres occurs, and by this means the motor and sensory projection tracts and the association fibres can all be differentiated from one another according to the time at which their myelinization takes place. (Figs. 9, 10.)

The *clinical* method consists in correlating the symptoms observed during life with the position of the lesion found after death. This method was used with particular success by Hughlings Jackson, who, from observations made on the distribution of paralysis and the occurrence of localized convulsions (Jacksonian epilepsy), was able to differentiate the functions of various parts of the motor cortex.

The *experimental* method, as practised by Hitzig, Ferrier, Horsley, Beevor, Sherrington, and others, consists in stimulating the cortex of an animal and observing the effects on different parts of the body. It has been particularly useful in enabling the motor area to be mapped out, and it is probable that the more recent results obtained by Sherrington and Grünbaum on the higher apes are for all practical purposes identical with the localization of the corresponding functions in man.

The results of *histological examinations* of the cortex, as carried out by Campbell and Brodman, show that the structure of different areas differs according to their function; while Bolton has made a valuable contribution to the subject in its pathological aspect by correlating the mental condition observed during life with the morbid histological appearances found after death.

Bolton divides the cortex into five primary layers:—

1. Outer-fibre lamina or superficial layer.
2. Outer-cell lamina or pyramidal layer.
3. Middle-cell lamina or granular layer.
4. Inner-fibre lamina or inner line of Baillarger.
5. Inner-cell lamina or polymorphic layer.

These layers, Bolton states, may be identified in all parts of the neopallium, but they differ in their degrees of development in different cortical areas, as instanced in the psycho-motor area, in which the third layer is of minimal depth.

By combining the knowledge obtained by these different methods it is possible to map out a considerable area of the cortex according to the functions exercised. (Fig. 11.)

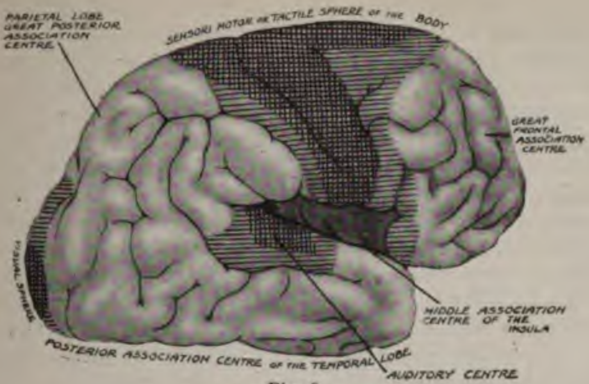

Fig. 9.

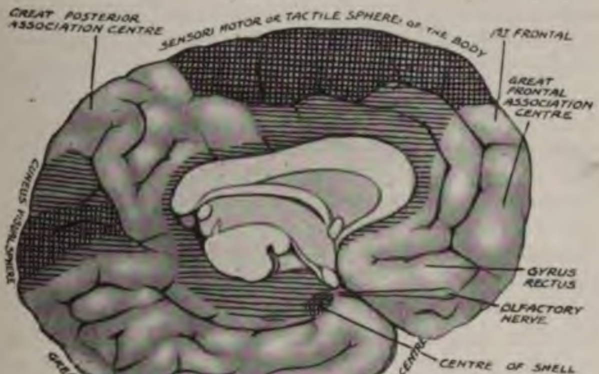

Fig. 10.

Figs. 9 and 10.—To show the projection and association centres of Flechsig, as seen on the external and internal surfaces of the right hemisphere. The dotted areas are the regions of the cortex which are first myelinized, because they constitute the arrival and departure platforms of the sensory and motor projection systems of fibres. The remainder of the cortex is myelinized at successively later periods, and corresponds to those regions of the brain which have been termed association centres. (*After Sir F. W. Mott, "Mental Development."*)

cortical area through which the initiat cesses appear to regulate the resulting when this part of the cortex is diseased there is apt to occur a condition known characteristics of which are that the pati he wants to do but is unable to do it, is no obvious paralysis of the part he For instance, when told to put out his to quite unable to do so, although immediatel may put it out quite naturally, as to mois such a case the mental initiation is appare so also is the motor projection, since mov carried out for other purposes, and the appears to lie in the mechanism throu psychic processes initiate the impulses f The mechanism for this process is situat terior parts of the *superior and middle* the left side, and communications are es the corresponding area of the right hemis ciation fibres passing through the corpus

At the foot of the *third frontal conv* left side, i.e. a little below the areas just de is Broca's convolution, which is closely a the motor mechanism for speech. There frontal region an area concerned with the eyes.

Immediately behind

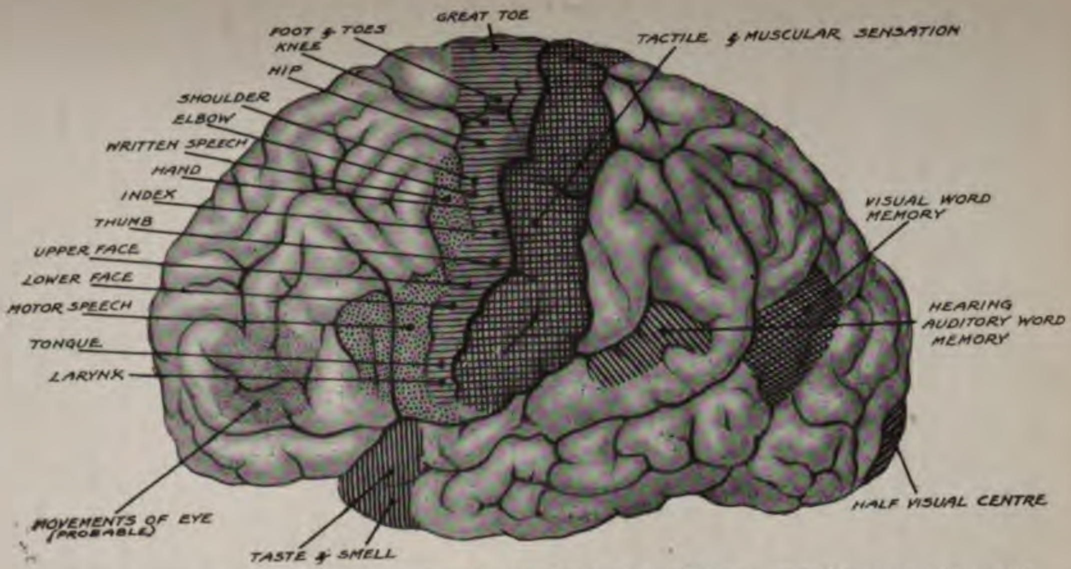

Fig. 11.—Lateral view of the left hemisphere, to show localization of the motor and sensory spheres and speech centres. (*After Sir F. W. Mott, "Mental Development."*)

The *prefrontal* lobes are especially associated with performance of the higher mental functions, and according to some observers the left lobe is more important in this respect than the right. This area of the cortex seems to be that in which the mental processes initiating or inhibiting the movements of the individual take their final form, and immediately adjoining it posteriorly is the cortical area through which the initiating mental processes appear to regulate the resulting movements, for when this part of the cortex is diseased on the left side there is apt to occur a condition known as *apraxia*, the characteristics of which are that the patient knows what he wants to do but is unable to do it, although there is no obvious paralysis of the part he desires to use. For instance, when told to put out his tongue he may be quite unable to do so, although immediately afterwards he may put it out quite naturally, as to moisten his lips. In such a case the mental initiation is apparently good, and so also is the motor projection, since movements can be carried out for other purposes, and the fault therefore appears to lie in the mechanism through which the psychic processes initiate the impulses for movement. The mechanism for this process is situated in the posterior parts of the *superior and middle frontal lobes* of the left side, and communications are established with the corresponding area of the right hemisphere by association fibres passing through the corpus callosum.

At the foot of the *third frontal convolution* on the left side, i.e. a little below the areas just described above, is Broca's convolution, which is closely associated with the motor mechanism for speech. There is also in the frontal region an area concerned with movement of the eyes.

Immediately behind the frontal area comes the *precentral* or *ascending frontal convolution*. This is commonly known as the motor area, and its cortex is characterized by containing the large Betz cells from which arise the pyramidal fibres that form the main paths for the transmission of voluntary motor impulses,

3. Connections are established with the cerebral cortex by fibres which form a further relay of the sensory tracts, and also by the fibres which conduct impulses from the cortex to the thalamus. It seems to be through these latter that the cortex exercises control over thalamic activity.

4. Descending fibres pass downwards to the cord in company with the rubro-spinal tract.

The functions of the thalamus have been investigated by Head and Holmes, who consider that it is the centre of consciousness for certain elements of sensation, and that it responds to all stimuli capable of evoking either pleasure, discomfort, or consciousness of a change in state.

The characteristic symptoms of a lesion of the thalamus are hemianæsthesia, spontaneous pains, and excessive reaction to painful stimuli on the opposite side of the body, with which may be associated involuntary movements of the affected limbs. Paralysis, though not resulting from a pure thalamic lesion, often accompanies it owing to the proximity of the motor tract. The activity of the thalamus appears to be controlled normally by the cerebral cortex, and when the communicating fibres through which this control is exercised are destroyed, over-activity of the thalamus results. This excessive activity shows itself by the different manner in which the two sides of the body react to disagreeable sensations, such as the prick of a pin or extremes of temperature, and to emotions. Music, for instance, may produce different reactions on the two sides, and Head and Holmes mention the case of one patient who was unable to go to his place of worship because he "could not stand the hymns on his affected side."

The causation of the "spontaneous" pains which are apt to occur in thalamic lesions is generally ascribed to irritation of the sensory fibres, but this explanation is not altogether satisfactory, and Holmes* has as described on p. 6. According to Sherrington and Grünbaum, the motor function, though dipping into the fissure of Rolando, does not extend, as formerly thought, on to the cortex of the ascending parietal lobe, which latter is apparently entirely concerned with the reception of sensory impulses. The remainder of the parietal lobe appears also to be concerned with sensory impulses.

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* *Trans. Seventeenth International Congress of Medicine*, 1913.

The *occipital lobe* is closely associated with vision, and two areas—the visuo-sensory, a lower projection area occupying the cortex of the cuneate lobe, and a higher centre, the visuo-psychic, occupying the area of the angular gyrus—can be recognized.

These two areas can be distinguished from each other by their histological appearances (Bolton), and the angular gyrus has important associations in connection with the visuo-psychic processes of language and recognition of objects, its destruction resulting in such conditions as alexia and word-blindness.

The cortex of the *temporal lobe* is concerned with hearing, and a lesion in this area will produce deafness of the opposite side, usually, however, only of a temporary character, owing to the cochlear nerves being connected with both sides of the brain. In the posterior part of the first temporal convolution on the left side is situated the auditory mechanism of speech, and disturbance of this area gives rise to various degrees of auditory aphasia.

Sensations of smell and taste appear to be located in the cortex of the *uncinate convolution*. (Fig. 11.)

The **optic thalamus** consists of an oval mass of grey matter situated at the base of each cerebral hemisphere just above the crura cerebri. The principal connections of each thalamus are as follows:—

1. The posterior part, known as the pulvinar, is continuous with the fibres of the optic tract.
2. It serves as a station for the reception of the main sensory tracts, which, having passed up from the cord, crossed in the medulla, and formed the median fillet, terminate in the thalamus (*see* p. 22).

3. Connections are established with the cerebral cortex by fibres which form a further relay of the sensory tracts, and also by the fibres which conduct impulses from the cortex to the thalamus. It seems to be through these latter that the cortex exercises control over thalamic activity.

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The causation of the "spontaneous" pains which are apt to occur in thalamic lesions is generally ascribed to irritation of the sensory fibres, but this explanation is not altogether satisfactory, and Holmes* has suggested that these so-called spontaneous pains are largely due to action of peripheral stimuli on an over-sensitive thalamus which has been deprived of the control usually exercised over it by the cortex. This suggestion, if correct, has an important bearing in therapeutics, since it should be possible to relieve the pains by diminishing as far as possible the peripheral stimuli that are likely to cause them. The origin of the involuntary movements which may be associated with thalamic disease is not quite clear, but they are thought to be due to disturbance of the functions of the red nucleus from which arises the rubro-spinal tract.

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* *Trans. Seventeenth International Congress of Medicine*, 1913.

Important investigations on the higher functions of the nervous system as a mechanism for the adaptation of the organism to changing external circumstances have been made by Pawlow.*

In his experimental work Pawlow observed that if external stimuli are applied simultaneously with the introduction of some acid substance into a dog's mouth, such external stimuli become in the course of time intimately connected with the salivary reaction which the acid sets up in the dog's mouth, i.e. a new reflex has been formed, the afferent impulses of which are produced by the external stimuli mentioned above.

To this newly formed type of reaction Pawlow gives the term **conditional reflex,** because of the number of conditions on which its formation depends. In order to produce such a conditional reflex it is essential that the indifferent stimulus should be applied synchronously with the introduction of the food or acid into the mouth, when, after a few sittings, it will be found that the stimulus has by itself become capable of arousing a secretion of saliva, and a "conditional reflex" has thus become formed. Pawlow concludes that the cerebrum is the organ by which sensations are analysed and through which new reflexes and new connections are constructed, and that it is by such means that the animal is enabled to adjust itself to the varied combinations of changes that are continually taking place in its surroundings.

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* *Brit. Med. Journ.*, Oct. 18, 1913, p. 973.

II. The Cerebellum

The cerebellum consists of a median lobe, known as the vermis, and of two lateral lobes.

To the naked eye each lobe is seen on section to consist of cortex, white matter, and central nuclei, of which latter the largest is the nucleus dentatum, others being the nuclei fastigii, globulosus, and emboliformis.

The **connections between the cerebellum and other parts of the central nervous system** are both numerous and important.

Among the principal of these are—

A. **Afferent fibres.**—1. Fibres entering the cerebellum from the cord by the direct cerebellar tract through the inferior cerebellar peduncles and by the tract of Gowers through the superior peduncles. The origin and course of these fibres have already been described (p. 18). Some fibres from the columns of Burdach and Goll also appear to enter the cerebellum, and an important connection with the posterior columns is established through the external part of the nucleus of Burdach, known as the nucleus of Monakow.

2. Communications from the nuclei of the pons, chiefly of the opposite side. The fibres establishing this communication form the middle peduncles.

B. **Efferent fibres.**—1. Fibres pass from the lateral lobes through the superior peduncles to the red nucleus and optic thalamus of the opposite side and also to the nuclei of the pons. Through these pontine nuclei, communications are established with the fronto-parietal and temporal regions of the cerebral cortex.

C. **Communications with the vestibular nucleus.**—The vestibular nerve is closely associated with the cerebellum through connections between the vestibular nucleus, the nuclei of the cerebellum, and the nucleus of Deiters. Communications are also established through the nucleus of Deiters with the ocular nuclei, as described under the head of Nystagmus (p. 93). Fibres likewise pass from the vestibular nucleus downwards to the cord by the vestibulo-spinal tract, where they establish communications with the cells of the anterior horns, and so form a mechanism which assists in maintaining muscle tone.

D. **Association fibres.**—By means of association fibres communications are established between different parts of the cerebellum.

Functions.—The cerebellum is principally concerned with the maintenance of equilibrium and muscle tone.

Impulses, which vary according to the position of the head in relation to surrounding space, are conveyed along the vestibular nerves to the cerebellum, whence the movements of the head and eyes are appropriately controlled.

If this mechanism is disturbed, nystagmus and vertigo, which are such frequent accompaniments of disease of the cerebellum, are produced.

Interference with the efferent fibres, of which the principal are the vestibulo-spinal tract and those flowing from the cerebellum to the optic thalamus and the red nucleus, is followed by loss of muscle tone (hypotonus) chiefly on the same side of the body as the lesion.

There may also be a difficulty in executing alternating movements of a limb, such as pronation and supination of the arm, with rapidity, and to this difficulty the term *adiadochokinesis* has been applied.

Coördination of muscular movements (*synergia*) and the regulation of the strength of nervous impulses passing to the muscles (*eumetria*) are carried out by means of the cerebellum.

The most obvious symptom of cerebellar disease is ataxy. Cerebellar ataxy is most marked in the coarser movements of the trunk and limbs, with the result that the patient walks with a reeling gait that has been likened to that of a person endeavouring to maintain his equilibrium on the deck of a rolling ship, to which there is a further similarity in the sudden lurch that the cerebellar patient occasionally gives to one side—usually towards the side of the lesion—or backwards if it is the vermis that is diseased.

In the arms the ataxy, though less marked, is shown by the erratic manner in which an attempt is made to make some particular movement, such as placing the finger on the tip of the nose—an attempt that is also often accompanied by tremor of the limb.

Babinski has pointed out that the movements in cerebellar disease are in excess of what is required to accomplish their object, and that when an attempt is made to place the finger on the tip of the nose the movement does not cease at the right moment but is prolonged in such a way that the finger passes over the nose and strikes the jaw. In the same way a line drawn on a sheet of paper is prolonged beyond the point at which it was desired that it should stop. The ataxy of cerebellar disease is modified but little by closing the eyes; in this particular it differs from that of tabes.

To sum up, it may be said that the cerebellum forms the centre on which the impulses of deep sensibility converge, and from which impulses pass to regulate the tone of the muscles and the smoothness of their action; and thus, in the words of Sherrington, it may be said that " the cerebellum is the head ganglion of the proprioceptive system."

CHAPTER VIII

EXAMINATION OF THE HIGHER FUNCTIONS OF THE NERVOUS SYSTEM

WHERE the symptoms complained of concern the higher functions of the nervous system it is important to test the integrity of those parts of the brain which underlie these functions, and as far as possible to obtain some record of the manner in which they respond.

The advances which experimental psychology has made in recent years now render it possible to bring many of its methods into common clinical use, thereby increasing the interest and accuracy of the diagnosis of some of the vaguer forms of nervous diseases usually classed under the heading of " neuroses." The value of such tests lies in the correlation of the results with all the other symptoms observed in the case, and they should be looked upon in the nature of additional physical signs.

The subject is a large one, and space allows only a brief account to be given of a few of the methods which the author has found of practical value.

Reaction times.—Records for reaction times for touch, vision, and hearing may in appropriate cases be of considerable value. The interval which takes place between the stimulus and the reaction not only serves as a guide to the rapidity with which coördination of the particular movement is carried out, but also forms a record for comparison between the states of the patient at different times.

The accompanying diagrams show the visual reaction times taken with the same instrument under precisely similar conditions in (1) a normal person, (2) an early stage of paralysis agitans, and (3) a late stage of paralysis agitans. (Fig. 12.)

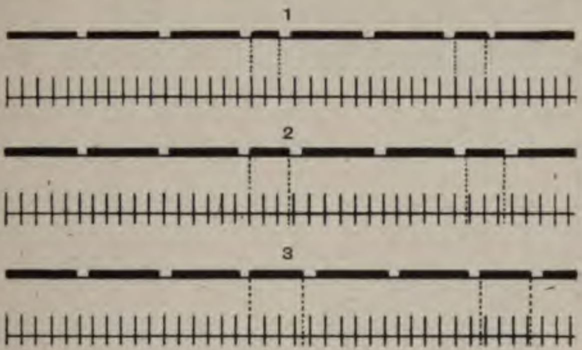

Fig. 12.—Records of visual reaction times in (1) a normal person, (2) an early stage and (3) a late stage of paralysis agitans. The vertical lines at the foot of each diagram are those of the time-marker, beating half-seconds.

Slowness of movements is a well-known symptom of paralysis agitans, and in Fig. 12 (2), where the patient was in quite an early stage of the disease, the record served as an additional corroboration of the diagnosis. Distinct abnormalities in reaction times, provided the afferent and efferent paths are normal, indicate the probability of faulty coördination of impulses in the higher cerebral centres; as such, when taken with other signs, they are suggestive of the presence of organic as opposed to functional changes, and thus may give a certain amount of assistance in diagnosing degenerations in their early stages.

Indications of **fatigue** may be recorded either by special instruments or by timing the quantity and estimating the quality of mental and physical work of a known standard.

A useful mental test for clinical purposes is that known as **time discrimination,** when given to those who are

8	6	3	7	7	9	3	5	6	[illegible]
9	4	6	8	9	4	7	9	7	8
5	9	8	5	3	2	8	6	9	8
7	2	8	7	6	4	3	8	5	8
2	5	7	2	8	2	8	7	9	4
6	2	8	3	7	9	7	5	3	5
7	9	4	9	2	2	4	8	4	2
4	5	7	6	3	9	3	6	5	5
7	2	6	2	9	9	4	2	9	7
2	8	3	5	6	4	7	8	7	2
2	6	6	5	9	4	5	8	8	3
8	2	6	2	3	3	4	4	6	2
4	5	2	5	7	5	4	6	4	3
5	9	6	2	9	5	6	6	4	9

The test

the approximately average time taken by ordinary persons.

Where this test is badly performed its repetition at a later date often gives a useful indication as to whether or not the patient has made any improvement. For instance, a neurasthenic patient who was accustomed to deal with figures in his daily work took sixty seconds over the test when first seen, but after a course of treatment, from which he derived great general benefit, he was able to reduce the time to forty-five seconds, thus corroborating the fact of his improvement.

Another useful form of test is that for **memory**. Patients are constantly complaining of loss of memory, and it is therefore important to be able to form some conclusion as to the reliability of their observations, since it very often happens that what is described as loss of memory is in reality due to feebleness of mental concentration, and not to any disturbance of the memory mechanism itself. Different types of memory may be tested, such as the *visual* (tested by saying off a series of letters or figures after having looked at them for a few moments), the *auditory* (tested by repeating a similar series that have been heard), and the *associative* (tested by reading out to the patient a list of words associated with one another, and then reading out the first words on the list and asking him to recall the associated ones).

Many other tests—e.g. for memory of time, place, and names—can be applied as required, and it is important to remember that the memory for recent events may in some circumstances be much weaker than that for events that happened long ago, as is frequently met with in the failure of memory that commonly occurs in old age.

These various forms of loss of memory may be associated with very different conditions. For example, an imbecile may possess a good visual or auditory memory and yet be unable to associate ideas to any appreciable extent; and the frequency with which memory for recent impressions fails before that for those of long standing has just been mentioned. As illustrating the interest of such tests, a patient complained, among other symptoms, that he was unable to remember the drift of speeches which he was in the habit of making, and on testing his memory it was found that while his visual and auditory memories were both good, his powers of remembering associations were weak. Here the patient's own account was confirmed by the test, and a more accurate estimation of the importance of the symptom could thus be made.

Jung's **word-association tests** are often of use as an aid towards discovering the particular line of thought with which the patient is troubled. This method consists in reading out a list of chosen words to the patient, instructing him to respond by uttering the first word which the read-out word suggests to him, while the examiner notes with a stop-watch the exact time taken in making the association. Jung has shown that this process of making associations is concerned with the emotions rather than with the intellect, and when one of the test words read out strikes some special mental complex the emotion thereby aroused is betrayed by certain peculiarities in the mode of reaction, and especially in the increased time taken to produce it. By observing the kind of words which have produced abnormal reactions, and by making up fresh test lists to include words especially connected with those that have already aroused emotion, it is often possible to derive assistance in coming to some conclusion regarding the complexes that are influencing the patient's actions.

Long reaction times and other peculiarities show an imperfect adaptation of the individual to the conditions the stimulus words have called up, thus indicating directions in which faulty reaction may take place. The method may be used also as a guide to progress by comparing the reaction types from time to time during the course of the illness.

The **psycho-galvanic reflex,** in which deflection of a galvanometer is produced by emotional states, can also be used to indicate the presence of emotional memories.

Degrees of mental deficiency can be estimated by the Binet-Simon tests and their modifications, for accounts of which the reader is recommended to consult special works.

CHAPTER IX

PATHS OF INFECTION OF THE CENTRAL NERVOUS SYSTEM

APART from extension by direct continuity of tissues, toxic material reaches the central nervous system principally by two channels—(1) the blood, and (2) the lymph.

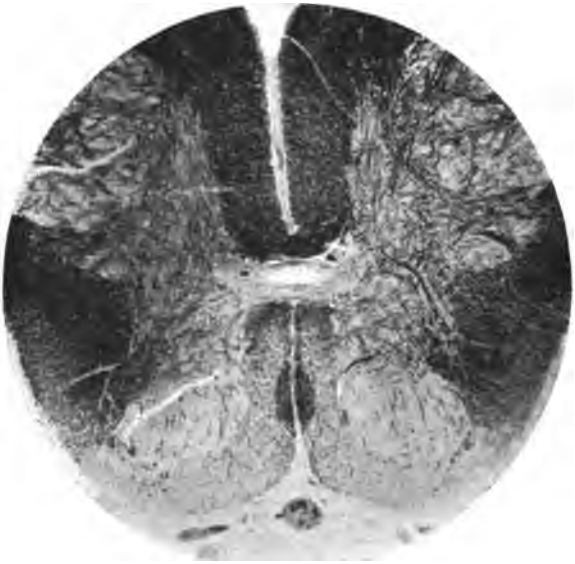

Fig. 13.—A lymphogenous infection—chronic tabes dorsalis.
(*Orr and Rows.*)

That infection may be hæmatogenous in origin has been long known, but the fact that it may also be lymphogenous depends upon later observations, and more especially upon the researches of Orr and Rows.*

The experimental work of these observers shows that infection may be conveyed to the cord through the lymphatic system of the peripheral nerves; and that this also happens in man has been proved by clinical observations in which infection of the cord has been traced by this path from such local conditions as carcinoma of the tongue, erysipelas of the face, bedsores in juvenile general paralysis, and tuberculous lumbar abscess.

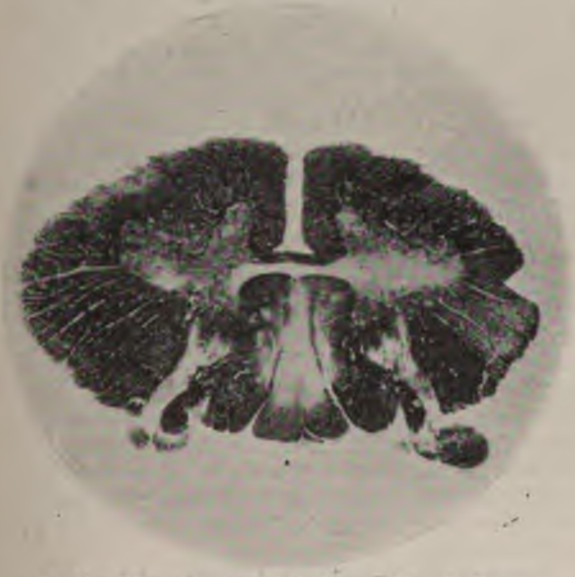

Fig. 14.—A hæmatogenous infection—Addison's disease.
(Orr and Rows.)

The authors also allude to a very important class of cases in which infection of the cord sometimes follows inflammation of the urinary bladder. They relate an instance in which transverse myelitis followed chronic bladder trouble, and refer to other instances of a similar nature.

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* *Proc. Roy. Soc. Med.* (Neurological Section), 1914.

From their clinical observations Orr and Rows conclude (1) that the locality of the lesion in the spinal cord always corresponds to the nerve supply of the infective focus; (2) that the degeneration of the intramedullary portion of the spinal roots commences at the point where the neurilemma sheath is lost; (3) that the posterior root entry zone is always most affected; (4) that, as examination of the extramedullary portion of the nerves yielded a negative result, it seems correct to assume that toxins can in certain cases ascend along the perineural lymphatics without producing parenchymatous changes in the nerves.

From the correlation of the changes found after death with the symptoms observed during life it appears that absorption may take place along the ascending lymph paths of the nerves for a considerable period before the exciting agent is of sufficient potency to cause symptoms. The explanation of this probably lies in the fact that the anatomical arrangement of the structures is capable of causing attenuation of the noxious substances as they approach the central nervous system.

The highly vascular epidural tissue and the dura itself form a barrier to the spread of infection. Thus in the early stages of infection the exciting agent reaches the cord in small quantities or in an attenuated condition and gives rise to little disturbance, but should the defence mechanism be broken down acute inflammation occurs.

Between the experimental results of lymphogenous and hæmatogenous infection Orr and Rows make the following distinctions:

In *lymphogenous infection* there are found (1) reaction of the cells of the fixed connective tissues; (2) proliferation of the cells of the adventitial sheath of the veins and capillaries; (3) the appearance of scavenger cells where the myelin is disintegrated; (4) nerve-cell degeneration and neuronophage phenomena.

In *hæmatogenous infection* (1) the most highly developed structures, i.e. the nerve cells, suffer least of all. (2) There is primary degeneration of the myelin sheath round the cord margin and on either side of the postero-median septum. (3) The myelin degeneration is greatest in the upper part of the cord. (4) There is œdema of the cord. (5) The vessel walls are hyaline, and contain thrombi of the same nature.

The difference in the distribution of the degeneration in the two forms of infection is shown in the accompanying figures (Figs. 13, 14).

Among inflammations which are probably of lymphogenous origin Orr and Rows include many cases of acute and subacute meningomyelitis, acute poliomyelitis, tabes dorsalis, and general paralysis, while those of hæmatogenous origin comprise examples of subacute non-systemic lesions of the cord such as may be associated with conditions like pernicious anæmia and Addison's disease.

CHAPTER X

LUMBAR PUNCTURE

Technique.—For a lumbar puncture the interval between the 3rd and 4th or between the 4th and 5th lumbar vertebræ should be selected. (Fig. 15.) A line joining the highest points of the iliac crests passes through the 4th lumbar spine, so that the puncture should be made either above or below the line, midway between the spinous processes, and ½ in. or rather less from the middle line. It is scarcely necessary to say that the skin, the needle, and the hands of the operator should be thoroughly sterilized. The skin over the chosen spot should be frozen, but in operating on children a general anæsthetic is sometimes necessary.

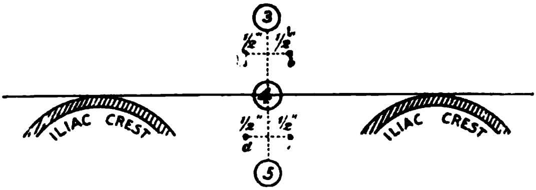

Fig. 15.—Diagram showing positions suitable for lumbar puncture. (*H. MacCormac.*)

The needle, which in the case of an adult should be about 3½ or 4 in. long, and in that of an infant about 2½ in., should have the calibre of an ordinary needle for the injection of antitoxin.

Position.—The patient should lie on the side, with the knees drawn up and the back arched outwards.

When all is ready, the needle is inserted forwards and slightly inwards, and if it strikes upon bone it must be withdrawn a little and its direction somewhat changed. There is no great difficulty in discriminating between bone and ligamentum subflavum, which also offers some resistance.

If the pressure is normal the fluid flows out drop by drop when the point of the needle is in the arachnoid space, but if the pressure is increased the flow is more forcible. Occasionally a little blood from some small vessel is mixed with the first few drops, but this soon disappears. Some operators prefer to use a syringe as a handle to the needle, but this is not necessary, and only in exceptional cases should any suction power be used to withdraw the fluid.

Value in diagnosis.—For ordinary purposes of diagnosis about a drachm of the fluid is sufficient, and it should be received into a sterilized test-tube. This may be chemically examined, and, after it has been centrifugalized, slides for the microscope* and cultivations of micro-organisms can be made.

Normal cerebro-spinal fluid is quite clear, and when examined under the microscope contains no cellular elements, or at most a very few lymphocytes.

In meningitis the fluid is often turbid, though in the tuberculous variety it may remain clear. In addition to bacteriological evidence of disease, microscopical examination shows the presence of lymphocytosis, and in the non-tuberculous varieties, such as epidemic cerebrospinal, streptococcal, and pneumococcal meningitis, polymorphonuclear cells also are generally present.

The reaction for sugar, which can be obtained from normal fluid, may be diminished or absent in meningitis.

Hæmorrhage of old standing into the cerebro-spinal fluid, from vessels of the meninges of the brain or of the cord, may cause a yellowish discoloration of the fluid, and in cases where the cord is compressed—especially towards its lower end—the fluid may show the characteristics known as Froin's syndrome—yellow discoloration, pronounced coagulability, and an increased number of lymphocytes.

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* For these Jenner's stain or methylene blue may be used.

Lymphocytosis can often be demonstrated in the early stages of general paralysis of the insane, tabes, and syphilitic diseases of the nervous system. It has also been found in disseminated sclerosis and anterior poliomyelitis.

Undue stress must not be laid on this method of diagnosis, and in all doubtful cases the results should be carefully considered in conjunction with the other signs before a decision is made.

Value in therapeutics.—The therapeutic value of lumbar puncture, apart from its use as a preliminary step to the injection of serums into the spinal canal, lies in the relief of intracranial pressure, though in most cases its effects are, unfortunately, only transient. It has, for instance, been found useful in cases of cerebral tumour and meningitis, and also in cases of coma from renal disease and injuries to the skull.

Some unpleasant symptoms, such as headache, nausea, and giddiness, may occasionally follow lumbar puncture, and it is always advisable for the patient to rest for some hours after the operation. Care must also be taken not to withdraw too much fluid; the amount naturally varies with the pressure of the fluid and the conditions for which it is drawn off, but if this point be borne in mind it is not likely that an excessive amount will be withdrawn.

SECTION II.—DISEASES OF THE PERIPHERAL NERVES

CHAPTER XI

PARALYSIS OF CRANIAL NERVES

I. THE FIRST OR OLFACTORY NERVE

The olfactory apparatus comprises the following elements: (1) The olfactory cells, situated in the olfactory mucous membrane, the central processes of which are continuous with the fibrils of the olfactory nerves proper. The latter end in (2) the olfactory bulbs, by arborizing round the termination of the dendrites of the so-called mitral cells, situated more deeply in the substance of the bulb. The centrally directed mitral axon is then conducted along (3) the olfactory tracts, which are each connected by means of a mesial and a lateral root with (4) the uncus, the cortical centre for smell. The olfactory apparatus thus consists of a series of neurones: the *olfactory cells*, the *mitral cells of the bulb*, and the *cortical cells* of the uncinate convolution.

The olfactory nerves proper, about twenty in number on each side, pass through the cribriform plate of the ethmoid, and tend to be arranged in an inner and an outer group; the fibrils of the former are distributed over the upper part of the ethmoidal septum, and those of the latter supply the mucous membrane overlying the superior turbinated bone. The *peripheral process* of the olfactory cells, of which these nerve fibrils are the central offshoots, ends on the surface of the olfactory mucous membrane, and is surrounded by a tuft of short filaments, the *olfactory hairs*.

To test the sense of smell, aromatic substances, e.g. cloves and peppermint, should be applied in turn to each nostril, the other being meanwhile closed. Substances which stimulate the 5th nerve, e.g. ammonia, must not be used, otherwise the sensations thereby produced may be mistaken for those of smell. The local causes of perversion or loss of this sense are very numerous, and must be excluded before the trouble can be attributed to the olfactory nervous apparatus. It should be remembered, too, that olfactory sensations may form the aura of epileptic fits, and may also occur in association with tumours of the uncinate convolution, which is the cortical centre for smell.

In hysteria also the sense of smell is frequently lost, usually on the same side as the hemianæsthesia, but occasionally the loss may be bilateral.

II. THE SECOND OR OPTIC NERVE, AND VISUAL PATHS

The optic nerve contains the visual fibres, and also serves as the afferent path of the pupil reflex to light.

Apart from disease of the structures of the eye itself, the sight may be impaired from lesions which interfere with the visual fibres at any part of their course. These may be grouped according as they are situated in—

1. The optic nerve.
2. The optic chiasma.
3. The fibres between the chiasma and cortex.
4. The cerebral cortex.

Anatomical (Fig. 16).—The **optic nerve** runs from the eyeball in front to the optic commissure behind, where a partial decussation of its fibres with those of the opposite side takes place. The nerve in its course traverses the orbit, the optic foramen, and the anterior fossa of the skull, and is invested with meningeal sheaths continuous with the dura and pia-arachnoid.

The nerve enters the globe of the eye at a point slightly below and on the inner side of the axis of the eyeball, and, after piercing the tunics of the latter, spreads out at the optic disc to form the innermost layer of the retina.

The **chiasma,** or **commissure,** situated at the base of the brain, is adherent to the floor of the 3rd ventricle, and rests upon the olivary eminence of the sphenoid bone; it is situated just anteriorly to the tuber cinereum, whilst at its sides the internal carotid arteries ascend to the brain.

At the commissure a partial decussation of the fibres of the two optic nerves takes place; thus, the fibres originating in the mesial half of each retina cross at the chiasma to join the optic tract of the opposite side, but those arising in the temporal portion of the retina do not cross the mesial plane, but are continued into the tract of the same side. The fibres which are situated in the posterior part of the chiasma are, in all probability, not connected with vision, but constitute a commissural bundle running on each side into the mesial root of the optic tract and connecting the two internal geniculate bodies together. This is known as the commissure of Gudden.

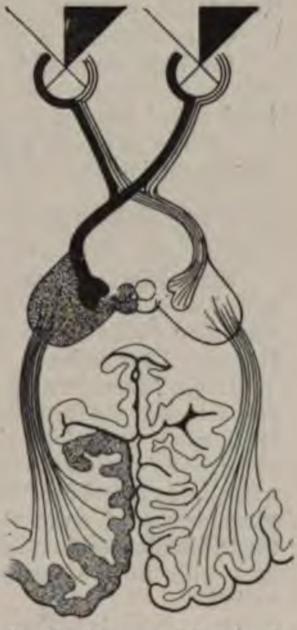

Fig. 16.—Diagram of the visual paths. (*After Dejerine.*)

The **optic tracts** proceed backwards around the crura cerebri, and each divides into a mesial and a lateral root. The mesial root has been referred to above, and is probably not connected with vision; the lateral root ends in the superior quadrigeminal body, the external geniculate body, and the pulvinar of the optic thalamus, which structures constitute the lower visual centres.

The cortical connections.—These lower visual centres are connected with the cerebral cortex by a large strand of fibres, known as the *optic radiation*. These fibres lie in the retroventricular part of the posterior limb of the internal capsule, and sweep backwards into the occipital lobe on the outer side of the posterior horn of the lateral ventricle.

Other connections of the lower visual centres are—

(*a*) With the nuclei of the ocular nerves—probably this connection is established through the posterior longitudinal bundle.

(*b*) The superior quadrigeminal body is connected by the mesial fillet with the medulla and cord.

1. Lesions of the Optic Nerve

OPTIC NEURITIS

Excluding local conditions in the eyeball itself, the causes of optic neuritis may be divided into two classes, according as they arise from (*a*) intracranial disease or (*b*) general toxæmias.

(*a*) In **intracranial disease,** meningitis, tumours, and abscesses are the commonest causes of optic neuritis. Quite rarely some faint signs of inflammation have been observed to precede the atrophy of disseminated sclerosis, tabes, and general paralysis of the insane. In meningitis some degree of optic neuritis frequently accompanies the tuberculous and streptococcal forms, but is less often met with in the cerebro-spinal and posterior basic varieties.

Optic neuritis is the most constant sign that accompanies cerebral tumours. It occurs in about 90 per cent. of the cases, and is of great diagnostic importance.

With abscess of the brain, neuritis, though frequent, is not so constant as in tumours, nor when present is it usually so intense.

Ophthalmoscopic appearances.—In the early stages the disc is hyperæmic and its borders are ill defined. As the process increases, the physiological cup, of which the normal appearance is shown in Plate 5, Fig. 1,

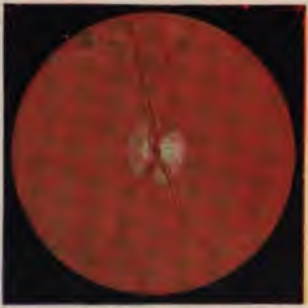

Fig. 1.—Average normal fundus; physiological cup.

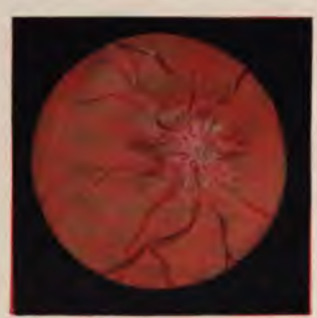

Fig. 2.—Optic neuritis, choked disc.

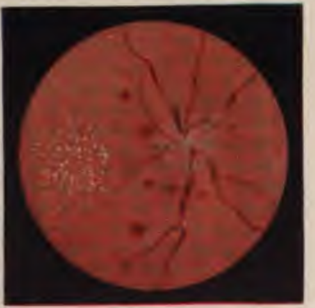

Fig. 3.—Albuminuric retinitis.

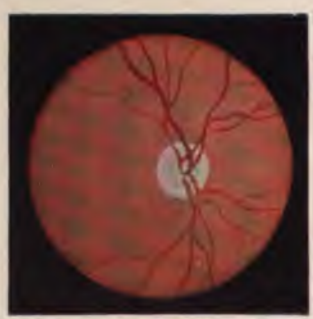

Fig 4.—Primary optic atrophy.

PLATE 5.—The fundus oculi in optic neuritis, albuminuric retinitis, and primary optic atrophy, compared with the average normal fundus.

(From Sym's "Diseases and Injuries of the Eye.")

becomes filled in and the course of the arteries at its edges obscured by inflammatory exudate. At the same time the disc becomes swollen (Plate 5, Fig. 2). Later on, changes appear in the retina. The veins are engorged and small capillary flame-shaped hæmorrhages take place into the retinal tissue. Lastly, the exudate is partially absorbed and partially organized into connective tissue; the optic nerve becomes white, the vessels small, and permanent failure of sight ensues.

It is important to note that in the early stages of optic neuritis the patient often notices little or no impairment of vision, and that systematic ophthalmoscopic and perimetric examination are the only methods which can be relied upon to detect its presence.

(*b*) **General toxæmias.**—The toxæmias associated with chronic renal disease, severe anæmias, diabetes, chronic lead-poisoning, and occasionally other infections, may all give rise to optic neuritis. (Plate 5, Fig. 3.)

RETROBULBAR NEURITIS

In retrobulbar neuritis there is an inflammation followed by degeneration of the axial fibres (i.e. the macular bundle). It is this condition that gives rise to the *toxic amblyopias* from tobacco and occasionally from other poisons.

Symptoms.—Vision is gradually impaired, especially for colours, so that the earliest signs are usually those of a central scotoma for green and red. For a time there may be no changes to be observed with the ophthalmoscope, but later there is some pallor of the disc, especially in its temporal half.

OPTIC ATROPHY

Optic atrophy may occur secondarily to the conditions enumerated under neuritis, or it may be due to a primary degeneration of the nerve fibres.

Primary optic atrophy is principally met with in tabes dorsalis and general paralysis of the insane, and in disseminated sclerosis. The toxic amblyopias, as stated above, are probably examples of axial neuritis.

In primary atrophy the disc slowly becomes paler, while the vessels undergo but little change. (Plate 5, Fig. 4.) Vision gradually fails, and the contraction of the colour field is at first generally greatest.

HEREDITARY OPTIC ATROPHY (LEBER'S DISEASE)

The condition—rarely met with—known as hereditary optic atrophy was first described by Leber. The disease, which is characterized by atrophy of the optic nerve, tends to run in families and is usually transmitted by the females and inherited by the males. It sometimes skips one generation, to appear again in the next. In the majority of cases the symptoms first appear in young adults (age from 15 to 20), but they have been observed to begin both in children and in elderly people.

The patients complain of failure of sight, and the ophthalmoscopic appearances are those of partial atrophy, preceded, possibly, by some slight signs of neuritis. The morbid changes occur in both eyes, but in most of the cases they do not progress far enough to cause complete blindness. The symptoms closely resemble those caused by tobacco, but in Leber's disease there is a central scotoma for white, whereas in tobacco amblyopia the failure of central vision is chiefly for red and green.

It seems probable that the pathological changes are very similar to those of tobacco amblyopia, and that the condition is in reality one of axial neuritis (Nettleship).

There is no adequate form of treatment known.

2. Lesions of the Chiasma

The chiasma is liable to be damaged by tumours at the base of the brain, especially by those connected with the pituitary body; hence it often happens that patients with signs of acromegaly complain first of their vision. Reference to the diagram (Fig. 17) shows that the first effect most likely to be produced by pressure on the chiasma is impairment of the function of those fibres which go to the inner side of each retina and supply the temporal field of vision, while later on the extension of pressure will affect the whole trunk of one or both nerves. The symptoms will in such a case begin with *double temporal hemianopia,* and end in complete atrophy.

It is possible, though naturally rare, in the process of atrophy of the visual fibres at the commissure, or owing to the symmetrical position of two tumours, for the fibres proceeding to the outer half of each retina to be caught, while those going to the inner halves are left intact. In that case, the function of the outer half of each retina being lost, there will be blindness in both nasal fields, i.e. a *double nasal hemianopia.*

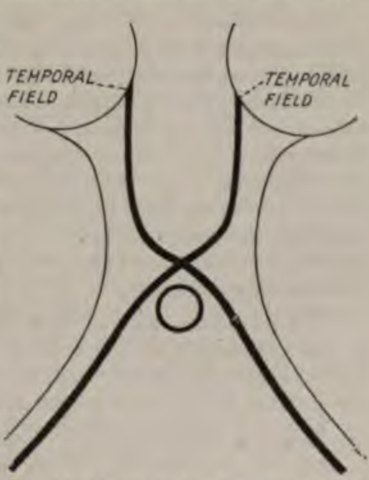

Fig. 17.—Diagram showing probable effects of pressure on the chiasma.

3. Lesions between the Chiasma and the Cortex

If the fibres are implicated anywhere in their course between the commissure and the cortex, e.g. in the neighbourhood of the internal capsule, then the outer part of one half of the retina and the inner part of the other will be affected—i.e. the temporal field on one side and the nasal field on the other—and thus a simple or *homonymous hemianopia,* which is by far the commonest variety, is produced.

4. Lesions of the Visual Cerebral Cortex

The area of cortex concerned with visual sensation lies chiefly on the mesial aspect of each occipital lobe, above and below the calcarine fissure.

Destruction of the visual area on one side or of the fibres passing from it (optic radiation) gives rise to an homonymous hemianopia of one or other side, according to the hemisphere affected. From observations made during the War by Gordon Holmes and Sir W. T. Lister, it appears that central vision is represented in the posterior part of the occipital areas and that a lesion at the tip of one occipital lobe (i.e. at the " occipital pole ") disturbs the central field of vision in both eyes by causing a small central scotoma immediately to one side of each fixation point.

The peripheral part of each half field is represented in the anterior part of the visual cortex, so that a unilateral lesion of this part will cause some constriction of both visual fields (so-called telescopic vision).

In some cases of injury to the visual areas not only is the field of vision disturbed, but there is, in addition, some loss of appreciation of depth and thickness in the objects viewed.

Wernicke's hemiopic pupil reaction.—This test is carried out by throwing a narrow beam of bright light on to the blind half of a hemiopic retina and noting if the pupil contracts.

Absence of any contraction is an indication that the lesion causing the hemianopia is on the peripheral side of the pupil centre, i.e. somewhere between the optic tract and the corpora quadrigemina; while the presence of contraction shows that the lesion is behind the centre for the pupil, i.e. in the optic radiation or occipital cortex.

The test is of some value as an aid in localizing cerebral tumours, in which hemianopia is one of the symptoms, but it is not an easy one to perform.

III. OCULAR NERVES

The 3rd, 4th, and 6th nerves may conveniently be considered in a group, since they all supply the muscles of the eye.

LESIONS OF THE THIRD NERVE

Anatomical.—The nucleus of this nerve is situated in the floor of the aqueduct of Sylvius. The fibres emerge on the surface of the inner side of the crus cerebri at the superior border of the pons, and, passing into the cavernous sinus, finally enter the orbit through the sphenoidal fissure and supply all the muscles of the eyeball except the superior oblique (4th) and the external rectus (6th). This nerve also innervates the levator palpebræ, the ciliary muscle, and the sphincter of the iris. The nerve enters the orbit in two divisions: the superior division supplies the levator palpebræ and superior rectus, and the inferior is distributed to the other muscles.

The dilator muscle of the iris obtains its nerve supply from the sympathetic.

The whole of the 3rd nerve or any of its branches may be affected. When there is **complete paralysis** of the nerve the upper lid drops (ptosis) from the loss of power of the levator palpebræ, and the effort that is made to keep the eye open is often reflected in the furrowed forehead produced by the contraction of the occipito-frontalis.

The eyeball is pulled outwards by the unantagonized external rectus, and the power of moving it inwards, upwards, and downwards is also lost; all that can be managed is a slight movement downwards and inwards by means of the superior oblique. The pupil is dilated and unable to react to light or during accommodation.

When, as often happens, there is only **partial paralysis** of the nerve, one or more of the muscles which it supplies suffer.

In paralysis of the **superior rectus** the upward and internal rotatory movements of the eyeball are impaired. The resulting diplopia is crossed, and the false image is above and tilted away from the true one. When the **inferior rectus** is paralysed the movements of depression and external rotation are impaired. The diplopia is crossed, and the false image is tilted towards the true one.

In paralysis of the **inferior oblique** the upward and external rotatory movements are weakened. The diplopia is homonymous, the false image being above and tilted away from the true one.

Congenital ptosis is sometimes met with, and is probably due to maldevelopment of the levator palpebræ. It is often associated with a deficient power of moving the eye upwards.

Another class of cases, also of congenital origin, is that known as "jaw-winkers." In these there appears to be a deficiency in the nervous innervation of the levator palpebræ, while at the same time there is an association of movement between it and the muscles of mastication. The result is that of ptosis while the jaw muscles are at rest, and an opening and closing of the eyes when masticatory movements are going on.

LESIONS OF THE FOURTH OR TROCHLEAR NERVE

Anatomical.—The nucleus of the 4th nerve is situated in the floor of the aqueduct of Sylvius, just behind that of the 3rd, and the fibres arising from it decussate in the superior medullary velum with their fellows of the opposite side. On emerging at the surface the nerve winds round the crus cerebri, lying between the posterior cerebral and superior cerebellar arteries, and, after passing through the cavernous sinus, enters the orbit through the sphenoidal fissure to supply the superior oblique, which moves the eyeball downwards and outwards with a wheel-like rotatory movement inwards.

Defective movement from weakness of this nerve is not easy to observe. The chief symptom is diplopia with giddiness on attempting to look downwards, as in coming downstairs.

The diplopia is homonymous, and the false image is on a lower level and obliquely inclined towards the true image.

LESIONS OF THE SIXTH NERVE, ABDUCENS

Anatomical.—The nucleus is in the floor of the 4th ventricle, on a level with the lower part of the pons. The fibres emerge from the groove between the pons and the medulla. Passing through the cavernous sinus, the nerve enters the orbit through the sphenoidal fissure, between the two heads of the external rectus, and terminates in this muscle.

In **paralysis** of this nerve there is deficient movement of the eyeball outwards, and when the loss of power is complete the eyeball cannot be moved beyond the middle line.

There is internal strabismus owing to over-action of the internal rectus, which in long-standing cases may become permanently contracted. There is diplopia on attempting to look outwards on the paralysed side. The diplopia is homonymous, and the true and false images are parallel.

If there is a lesion of the nucleus of the 6th nerve there will be paralysis of the internal rectus of the opposite eye, since its movements are governed through fibres from the nucleus of the 6th.

The general effects of **paralysis of the ocular muscles** may be summed up as follows:

1. **Limitation of movement.**—When a muscle is paralysed the movement of the eyeball is limited in the direction in which the muscle acts. This can be shown by asking the patient to follow an object with his eyes, when the results of loss of power in one or more directions will usually be manifest.

2. **Squint.**—When one or more muscles are paralysed, not only is there limitation of movement in certain directions, but the balance of power when the eyeballs re at rest is also lost, and the contraction of the unopposed muscles causes the eyeballs to deviate so that

their axes cease to be parallel, thereby producing the condition known as *strabismus*, or *squint*.

3. **Diplopia.**—Double vision is the result of the strabismus. The eyeballs having ceased to be parallel, the images no longer fall on corresponding parts of the retinæ, and so two objects appear to be present. It is shown by making the patient follow an object in different directions, and then, as soon as the affected eye ceases to fix that object, two images will be seen. Double vision does, not, however, necessarily accompany every case of squint, as the patient sometimes learns to disregard the false image.

When the false image is on the same side as the affected eye the condition is known as simple or *homonymous diplopia*, but when the images cross so that the one formed on the retina of the affected eye is projected in such a manner that it is opposite the sound eye the condition is known as *crossed diplopia*.

In all cases of internal squint—convergent strabismus—the diplopia is simple. If, for instance, the external rectus of the left eye is paralysed, the image will fall upon the retina to the inside of the yellow spot, and in the field of vision it will appear to the left of the image belonging to the right eye.

In all cases of external squint—divergent strabismus—the diplopia is crossed. Thus, when the axes of the eyes are prolonged forwards and the lines intersect the diplopia is simple, and when they diverge the diplopia is crossed.

In cases of paralysis of the internal and external recti the true and the false images will be parallel to one another, but in paralysis of the other muscles rotation of the eyeball comes into play and the false images become tilted.

4. **Secondary deviation** is an over-action of the sound eye, and is demonstrated as follows: If the sound eye is covered up, and the patient made to "fix" an object with the paralysed eye in the direction of the weak muscle, the eyeball turns as far as it can in the de-

sired direction, and at the same time the sound eye moves over a still greater distance in the same direction.

If a man has a weak external rectus on the left side, and his sound eye be covered up, and he is made to fix an object towards his left-hand side with the left eye, the latter will then attempt to turn as far as it can with its weak external rectus, and if the sound eye is watched it also will be seen to turn in the same direction, but the balance between them is upset, so that, instead of the two remaining parallel, the sound eye rotates through a larger arc than the weak one. This is due to the fact that the left external rectus and the right internal rectus always act together; both receive the same amount of stimulus to look to the left, but on account of the weakness of the external rectus on the left side the stimulus, in order to effect the movement, must be stronger than is usually necessary, and the internal rectus of the right eye, being sound, responds to this extra stimulus, and so overacts and moves the eyeball over a distance which is out of proportion to that of the left.

5. **Erroneous projection of the field of vision.**—This symptom depends on the limitation of movement of the eyeball, so that objects appear in one spot when they are in reality in another. It is due to the fact that the relations between objects and ourselves are judged by the range of movement of our eyes, and when this range of movement is suddenly upset the strong attempt which is made to move the eye in the direction of the weakened muscles occasions the idea that the eyeball has moved farther than it really has, and thus upsets calculations of distance which have been based on all previous experience.

If the external rectus on the left side be weak, then on turning the eye to the left there is an increased innervation of the weak muscle in order to try to produce the movement. As the patient has been in the habit of judging the amount of movement by the amount of innervation necessary to cause it, he naturally interprets this increased innervation as producing its full effect, and therefore he thinks his eye has turned farther to the left than it really has, and, as a consequence, objects appear to him to be farther to the left of himself than they really are.

NYSTAGMUS

Nystagmus is the term applied to oscillation of the eyeballs, which is usually involuntary, bilateral, and rhythmical. The movements most commonly occur in a horizontal direction (lateral nystagmus), and are generally manifested only on turning the eyes to the right or left, but they may be vertical or rotatory, and in some instances they occur while the eyeballs are at rest. Nystagmus can be produced physiologically in the following ways:—

1. The method of rotation which consists in turning a person round and round in a rotatory chair. The type of nystagmus (lateral, vertical, or rotatory) thus obtained depends upon the degree with which one semicircular canal is stimulated more than the others, and can be controlled to a large extent by the position in which the head is fixed during the rotation.
2. The caloric method, by which a stream of hot or cold water is gently run into the ear.
3. The passage of a galvanic current through the head.

All these methods produce nystagmus by disturbing the mechanism of the labyrinth, and they are used for testing the irritability and the presence of structural alterations of this organ.

Nystagmus is the result of a disturbance of the coördinating mechanism for movements of the eyeball, and such disturbances may be divided into three groups according as they arise from disorder of (*a*) the semicircular canals; (*b*) the eyes; (*c*) the central nervous system.

(*a*) The labyrinth, i.e. the semicircular canals, forms a peripheral organ from which impulses pass inwards to regulate the movements of the eyes, and disease or injury of the canals may be accompanied by nystagmus, such as occurs, for instance, in some cases of acute aural vertigo.

(*b*) The diseases of the eye which set up nystagmus are usually those occurring in childhood, such as various kinds of ophthalmia and keratitis, which prevent a clear formation of the image on the retina. Nystagmus is also apt to be associated with albinism.

(*c*) In the central nervous system nystagmus is caused by lesions which interfere with the paths along which coördinating impulses pass between the semicircular canals and the ocular nuclei.

From the semicircular canals the impulses are conducted along the vestibular nerve to the vestibular nucleus, and thence several communications are established, the principal of which are those with the nuclei of Deiters and Bechterew and the vermis of the cerebellum. It is probable that through the nucleus of Deiters connections are also formed with the dentate nucleus of the cerebellum. From the nucleus of Deiters, which is situated in the pons, communication is established through the fibres of the dorsal longitudinal bundle with the ocular nuclei. It thus follows that lesions which disturb this delicate mechanism of coördination are liable to cause nystagmus, and the principal nervous diseases in which such disturbance is apt to occur are disorders of the vestibular nerve, diseases of the cerebellum and pons, disseminated sclerosis, Friedreich's disease, and syringomyelia of the medulla and midbrain. Nystagmus has very occasionally been observed, likewise, in cases of alcoholic neuritis.

Nystagmus is also associated with the head-nodding of children known as spasmus nutans. Here the movements of the head, which often precede those of the eyes, generally begin during the first few months of life and tend to disappear when the child reaches the age of 2 or 3 years.

Cases in which nystagmus appears to be hereditary have been described.

Miner's nystagmus.—Nystagmus may arise in the course of various occupations, and is so prevalent in the case of miners that it is recognized as one of the industrial diseases for the contraction of which compensation may be claimed.

The cause of miner's nystagmus has been attributed by Snell to excessive muscular fatigue brought about by the position in which the miner works, but recent investigation seems to show that bad illumination with consequent imperfect formation of images on the retina is the principal factor, to which errors of refraction may probably be added as a contributory cause (Llewellyn).

Symptoms and diagnosis of nystagmus.—When nystagmus has existed from childhood there are often no symptoms, but when it arises in later life patients generally complain of the movements of objects.

So far as diseases of the nervous system are concerned, nystagmus may, generally speaking, be taken to indicate the presence of organic disease, and it is often particularly useful in aiding the differentiation of disseminated sclerosis from hysteria.

It must be distinguished from "nystagmoid" movements, which are jerks of the eyeball that lack the more even movement of nystagmus, and which, though they may often be regarded with suspicion, have not in themselves the same significance as the complete nystagmus.

IV. THE FIFTH OR TRIGEMINAL NERVE

Anatomical.—This nerve is partly motor and partly sensory in function. The **motor fibres** arise from the motor nucleus, which lies in the grey matter at the lateral angle of the 4th ventricle, and from the descending or mesencephalic root of the trigeminal, situated in the grey matter surrounding the Sylvian aqueduct. The small motor root passes forwards beneath the

Gasserian ganglion, and is wholly incorporated in the inferior maxillary division of the trigeminal. It innervates the temporal, masseter, the external and internal pterygoid, the mylohyoid and anterior belly of the digastric, possibly also the levator palati and azygos uvulæ (through Meckel's ganglion), and the tensor tympani and tensor palati muscles (through the otic ganglion).

Lesions of the motor root.—Lesions of this root are followed by loss of masticating power, and the lower jaw, when thrust forwards, is tilted to the weakened side by the unantagonized healthy muscles.

The **sensory root** of the 5th nerve ends in two nuclei of termination—(*a*) the sensory nucleus of the trigeminal, situated in the pons; and (*b*) the spinal root, connected with the substantia gelatinosa of Rolando.

The sensory root passes with its motor companion across the base of the brain and enters the Gasserian ganglion, which rests on the apex of the petrous portion of the temporal bone.

Thence the fibres separate into three great divisions (ophthalmic, superior and inferior maxillary), which supply sensation to the anterior portion of the skull, half of the face, including the orbit and eyeball, nose and nasal cavity, lips, teeth, mouth, and the anterior two-thirds of the tongue.

Lesions of the sensory root.—Neuralgia is very common in the area of distribution of one or more branches of this nerve. Division of the branches gives rise notably to anæsthesia, but often also to disturbances of nutrition of the skin, and especially of the eye—ulceration of the cornea and conjunctiva and suppuration of the whole eyeball being all possibilities. The most prominent trophic disturbances that occur over the area supplied by this nerve are, perhaps, those connected with facial hemiatrophy.

In spite of all these disorders of nutrition attending injury of the nerve, it does not necessarily follow that special trophic fibres exist. Many of the effects, especially those connected with the eye, can be accounted for by the loss of that protection against harmful stimuli which integrity of the sensory nervous system confers.

The 5th nerve makes many important connections, and the peripheral course of the fibres subserving the sense of taste is a somewhat complicated one. Two main descriptions of the course of these taste fibres are current:

1. It is held that the taste fibres from the anterior two-thirds of the tongue run at first in the lingual nerve, then in the chorda tympani, to the geniculate ganglion of the facial; subsequently by the great superficial petrosal nerve to Meckel's ganglion and the superior maxillary division of the 5th. The taste fibres from the posterior third of the tongue run at first in the glosso-pharyngeal as far as the petrous ganglion, then, by way of Jacobson's nerve, to the tympanic plexus, from which they follow the course of the small superficial petrosal nerve to the otic ganglion, and thus to the inframaxillary division of the trigeminal.

2. Edinger regards the nucleus of taste as a mass of grey matter, coextensive with the sensory nucleus of the 5th, which receives certain afferent fibres—(*a*) from the lingual nerve, through the medium of the Gasserian ganglion; (*b*) from the chorda tympani, through the geniculate ganglion of the facial, and the pars intermedia; and (*c*) from the glosso-pharyngeal, through the petrous ganglion (Box and Eccles).

FACIAL HEMIATROPHY

Facial hemiatrophy (Fig. 18) is a rare condition, and appears to depend upon a disturbance of nutrition in the area of distribution of the trigeminal nerve. It generally first shows itself during childhood, and is more commonly found in females than in males.

Symptoms.—The earliest signs are often found in the skin, which becomes shiny and pigmented with yellowish and brownish patches. The connective tissue soon takes part in the atrophy, and finally the growth of the whole side of the face, including the bones, is retarded. The muscles do not undergo any active atrophy, but they naturally waste from disuse and their movements are impeded by the condition of the tissues around.

The hairs of the eyebrows often turn grey or drop out, and the hair of the head on the diseased side is sometimes also affected. The cartilage of the ear is usually wasted, and the corresponding half of the tongue is sometimes atrophied, as seen in the illustration (Fig. 18); and here again it is the fat and connective tissue, not the muscles, that disappear. There may be deficient sweating on the affected side; the teeth are often brittle.

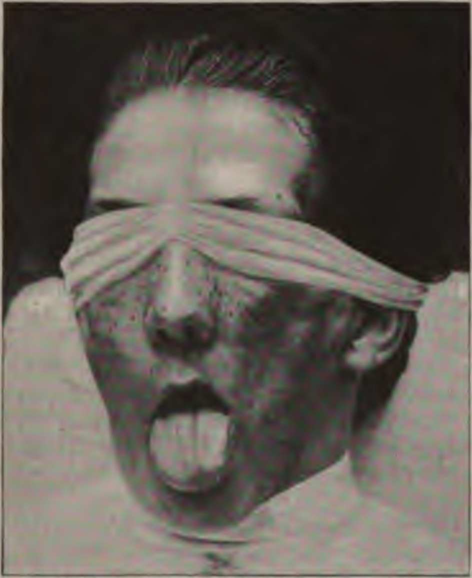

Fig. 18.—Hemiatrophy of the left side of the face and of the left half of the tongue.

There is usually no tendency for the atrophy to spread, though occasionally it extends to the structures on the other side of the face. There is very seldom any loss of sensation, but neuralgic pains may be felt over the wasted area.

Pathology.—The area to which the atrophy is generally confined has naturally led to a suspicion that some affection of the 5th nerve is at the root of the trouble. Sclerotic changes have been found in the nerve trunk, but it is difficult on this assumption to understand how the sensory fibres escape so completely.

The possibility of the atrophy being due to changes in the sympathetic has been suggested, but there is very little evidence to support it, except that dilatation of the pupil on the affected side has been noted; and this was well marked in the case illustrated in Fig. 18.

The most plausible idea is that which regards the condition as a tropho-neurosis due to a failure of the trophic function of the 5th nerve to the exclusion of its sensory and motor functions. This explanation, however, assumes the existence of special trophic fibres, of which there is no adequate proof.

Treatment.—The wasted parts cannot be restored. All that can be done is to maintain the nutrition, general and local, at as high a level as possible, and thereby to try to prevent the disease from extending.

V. THE SEVENTH OR FACIAL NERVE

Anatomical.—This nerve arises from a nucleus in the pons below the floor of the 4th ventricle. The fibres are at first in close relation with the nucleus of the 6th nerve, around which they wind. This fact is of value in the localization of cerebral lesions, since their proximity makes the two nerves very liable to suffer together from any gross disease in the neighbourhood of their nuclei.

The facial fibres appear at the surface of the brain at the angle between the pons and cerebellum, and, in company with the auditory nerve, but separated from it by the pars intermedia of Wrisberg, they pass across the base of the brain and enter the internal auditory meatus. Here the facial leaves the auditory nerve and enters the aqueductus Fallopii, through which it passes, and finally leaves the skull by the stylo-mastoid foramen to supply the muscles of one lateral half of the face and the platysma.

During its passage through the aqueductus Fallopii the nerve swells out into the geniculate ganglion, from which spring the greater, lesser, and external superficial petrosal nerves, and it also gives off, while passing through the canal, a branch to the stapedius and the chorda tympani, which last, leaving the main trunk a little above the stylo-mastoid foramen, turns forwards and, after passing through the tympanum and communicating with the otic ganglion, joins the lingual nerve, and so is conducted to the tongue to supply the anterior two-thirds with sensation of taste, the posterior third being supplied by fibres from the glosso-pharyngeal.

PERIPHERAL FACIAL PARALYSIS (BELL'S PALSY) *

Etiology.—The principal causes of peripheral facial palsy may be grouped according to the situation of the lesion.

1. **In the pons.**—The facial nucleus may be injured by tumours, or, more rarely, it may be the seat of an acute inflammatory process and form part of an acute poliomyelitis. Nuclear degenerations are sometimes met with in the course of cases of progressive bulbar paralysis, tabes, and disseminated sclerosis.

2. **At the base of the brain.**—As the facial nerve passes over the base of the brain to reach the internal auditory meatus it may again be compressed by tumours, especially by those which grow in the angle between the pons and the cerebellum. It is also apt to be injured by gummatous and other forms of meningitis, and by fractures of the base of the skull.

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* Strictly speaking, "Bell's palsy" includes only that form in which the lesion occurs after the nerve has left the stylo-mastoid foramen, but the term is frequently used for all forms of peripheral lesions.

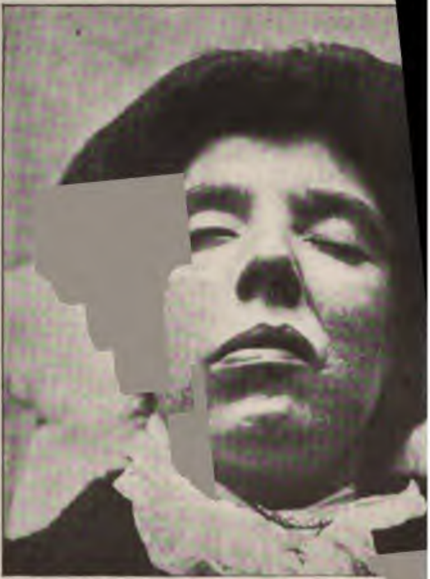

Fig. 19.—Right-sided facial paralysis of pe
Note the inability to close the eye and
tion of the naso-labial fold on the paral

4. Outside the canal.—Aft

more especially a draught such as may occur from the windows of a railway train. It is generally considered that inflammation of the nerve sheath is the primary lesion, and that the consequent exudation, having no room to expand in the bony canal, compresses the nerve fibres. If this hypothesis is correct these "rheumatic" cases may be regarded as being analogous to many cases of sciatica where an inflammation of the nerve sheath is also frequently the primary lesion; but it is, of course, possible that different toxins give rise to the paralysis in different cases, for the occasional extension of the paralysis to the facial nerve in a general neuritis (e.g. from alcohol or diphtheria), though rare, shows that the facial nerve is not immune from the poisons which are more apt to injure the nerves of other parts of the body.

Symptoms.—Peripheral lesions (i.e. lesions of the lower neurone situated at or below the nucleus) of the trunk of the facial nerve result in paralysis of the muscles supplied by that nerve, with some additional symptoms according to the locality of the lesion.

The paralysis being of the lower-neurone type, the loss of power is accompanied by wasting and changes in the electrical reactions. The paralysed side of the face looks somewhat flattened (Fig. 19); the nasal fold is obliterated, and the angle of the mouth drops, giving to the patient the idea that it is "drawn up" on the sound side; the nostril on the affected side is somewhat smaller. In some cases the paralysis is bilateral (Fig. 20).

1. **Loss of power.**—In a well-marked case there is loss of power of all the muscles on one side of the face, and this may be demonstrated by asking the patient to perform the following actions:

(*a*) *To look upwards.*—The forehead will remain smooth, owing to paralysis of the occipito-frontalis.

(*b*) *To frown.*—There will be no furrow, owing to loss of power in the corrugator supercilii.

(*c*) *To close the eyes.*—The eye on the affected side cannot be closed, and the attempt to close it is accompanied by a rolling upwards and inwards of the eyeball. This failure to close the eye is due to weakness of the orbicularis palpebrarum, and owing to the laxity of the fibres of this muscle in the lower lid the tears are apt to run down the face (epiphora). Conjunctivitis from irritation of the unclosed eye may also arise.

(*d*) *To show the teeth.*—The mouth does not move towards the paralysed side.

Other symptoms that may be noted are:

Weakness of the buccinator muscles, causing a tendency for the food to stagnate between the cheek and the gums.

Affections of hearing.—In some cases where the nerve to the stapedius has been paralysed, hearing has been noted to be more acute than usual, due, it is thought, to the unantagonized action of the tensor tympani tightening the drum.

2. **Loss of taste.**—If the chorda tympani is affected, taste will be lost over the anterior two-thirds of the tongue on the same side as the facial paralysis.

It is tested by placing a little bitter or sweet substance (e.g. quinine sulphate or sugar) on the side of the protruded tongue, and asking the patient to hold up his hand if he tastes anything. If he is allowed to speak or to move his tongue about, the substance is liable to be diffused and tasted on the opposite side and at the back of the tongue.

There is no loss of cutaneous sensation, but pain is sometimes experienced in the early stage of the paralysis.

Diagnosis.—There is usually no difficulty in determining whether facial palsy is present. The only condition which may perhaps resemble it superficially is that of *facial hemiatrophy*, but in the latter the muscles, though rigid, are still capable of voluntary contraction, and it is easy to see that changes in the skin and connective tissue are the main sources of the trouble.

Having decided that paralysis is present, it is necessary to consider whether the cause lies in the muscles, the lower neurone of the nerve, or the upper neurone.

The muscles of the face undergo primary degeneration in some of the myopathies, and weakness is frequently met with in cases of myasthenia gravis, but neither of these conditions need be confused with peripheral facial paralysis.

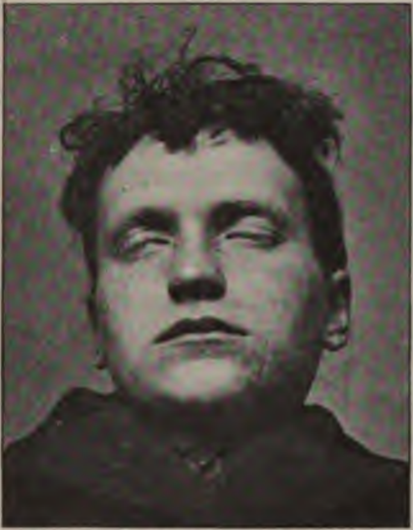

Fig. 20.—Bilateral facial paralysis. Note the inability to close the eyes and the general expressionless appearance of the face.

In the *myopathies* the muscles chiefly affected are those round the eyes and at the angle of the mouth; both sides are involved, and in addition there will nearly always be some signs of wasting of the trunk muscles.

In *myasthenia gravis* it is unlikely that only one half of the face would be weakened, and, further, the variation of the signs, together with the probability of ptosis and bulbar symptoms, makes it very unlikely that serious difficulty would arise in that direction.

A primary muscular origin of the weakness being thus excluded, the next point to be decided is whether the lesion is in the lower or the upper neurone, and this it is usually possible to do by noting the distribution of the weakness; for whereas in lesions of the lower neurone there is generally paralysis of the whole of one half of the face, in those of the upper neurone the movements which habitually take place together on the two sides (bilateral movements) escape or are only weakened. Thus, whereas in lower-neurone lesions the patient cannot close the eye, frown, or wrinkle the brow, in upper-neurone lesions all these actions can usually be performed. If, however, there is still any doubt as to which neurone is affected, it may usually be dispelled by an examination of the electrical reactions.

Having settled that the lesion is situated in the lower neurone, one must next try to determine its exact position.

1. If the facial paralysis is accompanied by paralysis of the external rectus on the same side, it suggests (provided there is no reason for thinking that there is more than one focus of disease present) that the lesion is at the facial nucleus itself, for it is there that the 6th nerve comes into close proximity to the 7th.

2. If deafness is present, it may be that both auditory and facial nerves are injured where they are situated close to each other at the base of the brain; but care must be taken before this view is accepted, for it may be that the deafness is due to middle-ear disease, and that the facial paralysis depends upon necrosis of the surrounding bone.

3. If taste is lost, it is evident that the disease is situated in that portion of the nerve wherein the chorda tympani runs, i.e. between the geniculate ganglion and a point a little above the stylo-mastoid foramen.

Prognosis.—The outlook in a case of peripheral facial paralysis must, of course, largely depend upon the cause. Those cases in which the neuritis is apparently of a "rheumatic" nature, and which appear to be caused by exposure to cold, for the most part do well.

In those which follow disease of the bone the result is more doubtful; while the prognosis in those produced by new growth or by complete severance of the nerve must depend upon the possibility of removing the tumour or of suturing the nerve trunk.

In the so-called "rheumatic" cases which so commonly come before us, an incomplete palsy is a favourable sign by showing that the intensity of the inflammation is not great, and such cases generally recover rapidly. When the palsy is complete the electrical reactions are a useful guide. If, in a week or ten days after the onset of the paralysis, reaction to the faradic current still persists, a steady return of power may generally be predicted; if, on the other hand, the paralysed muscles give a reaction of degeneration, recovery will be much slower and more uncertain; and, lastly, if all reaction to galvanism ceases, the outlook is still worse.

Late results of facial paralysis.—If complete recovery does not take place the paralysed muscles often gradually undergo some contraction, which at first sight has the effect of making the sound side appear to be paralysed, since the angle of the mouth on the contracted side is drawn up. This contraction is probably accounted for by changes in the muscle fibres, and about the time that it is beginning to take place a curious irritability is sometimes observed, whereby an attempt to contract one muscle is accompanied by the contraction of another which is not desired. For instance, on trying to close the eye, the corner of the mouth on the paralysed side may be simultaneously drawn up. It is as though there were one sheet of muscle instead of individual segments. This late contraction does not appear to take place in cases where the continuity of the nerve has been completely severed. Fibrillary tremors may also take place in the paralysed muscles.

Treatment.—The best method of treating the "rheumatic" cases is by regular faradization of the muscles; their nutrition is thus maintained so that they are ready to react to the impulses as soon as the latter are able to pass along the nerve; possibly the nutrition of the nerve-endings is improved at the same time. When there is no reaction to the faradic current it is customary to apply galvanism, but it is doubtful whether the latter gives any better results, even though the muscles react to it when they do not to faradism.

Internally, potassium iodide in doses of about 5 gr. seems to be useful; and, at the beginning, counter-irritation, in the shape of a blister behind the ear, sometimes appears to be of value.

Where the paralysis is due to necrosis of bone the treatment must still be conducted on the same lines, and in many cases the ultimate results are good, since the nerve is generally injured by extension of inflammation rather than by pressure of bone; and when the inflammatory process subsides, power returns, provided that the nutrition of the muscles has been maintained.

If at last it appears clear that the paralysis is permanent, the question of an operation may be raised, for it has been shown to be possible sometimes to restore a certain amount of movement by severing the facial nerve and grafting the end of the peripheral portion on to the spinal accessory or hypoglossal nerve. In most cases, however, the deformity and inconvenience produced by the facial paralysis are slight, and are preferable to the problematical benefits to be obtained from an operation which has its own very distinct risks.

Facial Spasm

Spasms of muscles, like paralyses, may be divided into three main groups according as they arise from a lesion of the lower neurone, the upper neurone, or one of "functional" origin.

(*a*) Of the **lower neurone** varieties of facial spasm mention has already been made of the muscular tremor which occasionally occurs as a sequela of peripheral facial paralysis (p. 106). There is also the "tic douloureux" which is a reflex spasm that sometimes accompanies severe trigeminal neuralgia, but which from its title must not be confused with the true tics which are of mental origin. Spasm may also be sometimes produced by irritation of the trunk of the facial nerve, e.g. by tumours, meningeal inflammation, or disease of bone.

Another form is a chronic fibrillary tremor which begins with a mere flickering of fibres of a muscle, often those of the orbicularis palpebrarum, and then gradually spreads over that half of the face. At first there may be long periods of intermittence, but later, as the condition progresses, the patient is hardly ever free. The spasms undoubtedly vary to some extent with the health of the patient, especially in the earlier stages of the trouble, but the disease is very intractable, and drugs, electrical methods, and massage are all usually powerless to produce any beneficial result. Injection of alcohol into the trunk of the facial nerve has been carried out in some cases as a means of arresting the spasm. The immediate result is facial paralysis with cessation of tremor, but the chief drawbacks to this method are (1) that a permanent paralysis may possibly be produced, and (2) that with the recovery of the nerve the tremor is likely to return. It is, therefore, a method to be practised only after careful consideration.

(*b*) In the **upper neurone** any irritation of cortical origin may produce a spasm, and the author recently had such a case under observation in which the earliest sign of a tumour of the facial area of the cortex was a slight paroxysmal spasm of the kind.

(*c*) Spasms of **functional** nature are of mental origin and come into the categories of hysteria and tics, which are considered elsewhere.

Occasionally chronic facial spasms are left as the result of chorea.

VI. THE EIGHTH OR AUDITORY NERVE

Anatomical.—The auditory nerve consists of two elements—(1) the cochlear nerve, (2) the vestibular nerve.

1. The **cochlear nerve,** on which is situated the ganglion spirale, divides directly into branches to (1) the macula acoustica of the saccule, (2) the ampulla of the posterior semicircular canal, (3) the organ of Corti in the cochlea. Centrally, the fine fibres of the cochlear nerve end in a ganglion in intimate relation with the restiform body, which may be described as consisting of two parts: (*a*) tuberculum acousticum, or lateral cochlear nucleus; (*b*) ventral cochlear nucleus.

Central connections of the cochlear nerve.—From the cells of these nuclei two tracts arise: a ventral tract, composed of the fibres of the corpus trapezoides, and a dorsal tract, represented by the striæ acousticæ. These tracts cross the middle line, decussating with the corresponding fibres of the opposite side, and then the strand turns upwards to become the lateral fillet; the fibres of the lateral fillet end in the inferior quadrigeminal body and the corpus geniculatum internum, and from the latter a tract proceeds to the cortex of the superior convolution of the temporal lobe. But it should be noted that certain internodes are situated in the corpus trapezoides and the lateral fillet, of which the most important is the superior olive, by means of which a communication is established with the nuclei of the 3rd, 4th, and 6th nerves. In this way the organ of hearing is brought into connection with the nuclei which preside over the movements of the eyeballs.

The succession of neurones which build up the sensory chain linking the organ of hearing to the cortex are therefore—

(1) Bipolar cells of the ganglion spirale.
(2) Neurones of the terminal cochlear nuclei;
(3) Neurones of the superior olive and nucleus of lateral fillet.
(4) Neurones of the corpus geniculatum internum.

2. The **vestibular nerve,** on which is the vestibular ganglion, enters the brain on the mesial side of the ventral cochlear nucleus; its fibres end in three nuclei of termination: (*a*) principal dorsal nucleus, (*b*) nucleus of Deiters, and (*c*) perhaps "nucleus of descending root."

Central connections of the vestibular nerve.—Their connection with the nucleus of Deiters brings the fibres of the vestibular nerve into relationship with other parts of the mechanism of equilibration, for Deiters' nucleus constitutes an internode in the path of those fibres which connect the cortex and roof nuclei of the cerebellum with the motor apparatus of the spinal cord; it also receives fibres through the vestibular nerve from the labyrinth of the ear, and sends fibres into the posterior longitudinal bundle, through which, in all probability, it influences the nuclei of the ocular nerves.

Lesions of the auditory nerve.—The chief symptoms of disorder of the auditory nerve are deafness, tinnitus, and vertigo (Chapter LIV.). The nerve is very liable to injury, often in conjunction with the facial, from the pressure of tumours, especially those growing at the cerebello-pontine angle, and it may also be damaged along with the facial in fractures of the base of the skull.

Atrophy of the auditory nerve sometimes occurs in locomotor ataxy.

VII. THE GLOSSO-PHARYNGEAL AND VAGUS NERVES

Anatomical.—The motor or efferent fibres spring from the dorsal motor nucleus and the nucleus ambiguus, lying deeply in the substance of the medulla; these fibres join the vagus and glosso-pharyngeal nerves.

The afferent fibres of the glosso-pharyngeal and vagus end in the dorsal nucleus and in the funiculus solitarius.

The glosso-pharyngeal or 9th nerve emerges from the upper part of the medulla oblongata, and leaves the cranium by the jugular foramen. It then appears in the interval between the internal jugular vein and the internal carotid artery, and is directed downwards and forwards to the tongue. It distributes branches to the mucous membrane of the tongue, pharynx, and middle ear, as well as to the stylo-pharyngeus and possibly to the middle constrictor of the pharynx.

The vagus or 10th nerve, which also leaves the skull by the jugular foramen, is directed downwards in the carotid sheath, and enters the thorax and abdomen. Each vagus is connected with the following cranial nerves: spinal accessory, glosso-pharyngeal, facial, and hypoglossal; also with some spinal nerves, and with the sympathetic in the neck, thorax, and abdomen.

Lesions of the glosso-pharyngeal.—The exact functions of this nerve are not known, owing to the rarity of cases of isolated paralysis, since it is intimately connected with the fibres of the vagus. Sense of taste may be impaired in the posterior third of the tongue, and there is some difficulty in swallowing, owing probably to paralysis of the stylo-pharyngeus.

Lesions of the vagus.—Affections of the nuclei in the medulla are accompanied by unilateral or bilateral palsies. Lesions of one nerve cause unilateral paralysis of the soft palate and larynx (probably through spinal accessory fibres), as is shown by the uvula being drawn over to the healthy side on phonation and by one vocal cord assuming a position nearly midway between abduction and adduction—the so-called "cadaveric position." The paralysed cord does not move either during phonation or respiration, but, on phonation, the healthy cord moves over the middle line to try to meet it. Owing to their length, one or other vagus nerve is particularly likely to be damaged, and the left recurrent laryngeal, which winds round the arch of the aorta, is especially apt to suffer from pressure of aneurysms. In this case the paralysed cord assumes the position described above; the voice is hoarse and weaker than usual, the cough is harsh, and the power of coughing is much diminished owing to the inability to bring the cords firmly together.

The fibres to the *abductors* of the vocal cords are more easily damaged than those to the adductors; hence in the earlier stages, before the paralysis is complete, the unantagonized adductors are apt to draw the cord to the middle line (Semon) till they, in their turn, lose their function, and the cord swings into its position of complete paralysis. During the stage in which one abductor only is affected there are no definite symptoms to draw the patient's attention to his throat; the movements of the healthy cord are sufficient for ordinary respiration, and, since the two cords can meet in the middle line, vocalization is not seriously interfered with. This stage of the paralysis is therefore likely to remain undiscovered unless a systematic examination of the larynx is made.

Both cords may be paralysed if the nuclei of the nerves are diseased, in which case the voice and the power to cough are both completely lost.

In paralysis of both abductors the cords come together in the middle line, and they do not separate during inspiration. The voice and power of coughing are but little affected, but there is obstruction to respiration, which shows itself by loud stridor.

If the abductor is paralysed on *one* side only, the cord on that side will approach the middle line, but there will be no definite symptoms so long as the movements of the other cord are freely carried out. This, as already mentioned, is apt to occur from pressure on the recurrent laryngeal before the adductors are paralysed.

In *adductor* paralysis the cords cannot be brought together, and the voice is lost. This form of paralysis is usually only partial, and is the form which commonly occurs in hysteria, when the cords cannot be voluntarily brought together for phonation, but usually move freely for coughing, and therefore the patient may be able to cough although the voice is lost.

Spasm of the muscles of the larynx may occur from central or peripheral irritation.

The result of general laryngeal spasm is closure of the glottis owing to the predominating power of the adductors. One of the commonest forms of laryngeal spasm occurs in rickety children, under the name of *laryngismus stridulus.* The attacks, which occur chiefly at night, are characterized by acute dyspnœa, which lasts for some seconds, followed by loud crowing inspirations as relaxation of the muscles takes place.

Laryngeal spasm occurs also in epilepsy and occasionally in hysteria. It may be induced, too, by local irritation, such as from foreign bodies or from tuberculous or syphilitic ulceration of the larynx.

The respiratory and cardiac rhythms may also be modified from disease of the vagus.

VIII. THE ELEVENTH OR SPINAL ACCESSORY NERVE

Anatomical.—The spinal accessory nerve consists of two divisions. The *spinal division* arises by a series of filaments from the cells of the anterior horns from the 1st to the 5th cervical segments, and passes upwards through the foramen magnum to join the accessory portion, in company with which it leaves the skull by the jugular foramen, and, after passing either in front of or behind the internal jugular vein and perforating the sterno-mastoid, crosses the posterior triangle of the neck, to end in the trapezius. It supplies this latter muscle as well as the sterno-mastoid.

The *accessory division* is to be regarded rather as an extension of the vagus. It arises from nuclei in continuation with those of the vagus, and, after passing out of the skull with the spinal portion, leaves it to return again to the vagus; and it ultimately supplies fibres to the pharyngeal and superior and recurrent laryngeal nerves.

The spinal accessory, like the vagus, is apt to be compressed by tumours and to have its nuclei affected in bulbar lesions.

The results of paralysis of the spinal portion are loss of power and wasting of the sterno-mastoid and upper part of the trapezius.

Paralysis of one sterno-mastoid gives rise to difficulty in rotating the head to the opposite side, and when power is lost in both there is a tendency for the head to fall back.

When the upper and middle fibres of the trapezius are paralysed the movements of shrugging the shoulders and raising the arm above the horizontal position are impaired, but the latter movement, though weakened, can still be accomplished by means of the serratus magnus.

When the lower fibres are paralysed the position of the scapula is altered, so that the vertebral border stands out prominently, as in paralysis of the serratus magnus, and there is also some loss of power to rotate the arm backwards.

The sterno-mastoid and trapezius often suffer together, but the nerve is sometimes affected after it has left the sterno-mastoid, in which case the trapezius suffers alone.

The sterno-mastoid and trapezius receive fibres also from the 2nd and from the 3rd and 4th cervical nerves respectively.

IX. THE TWELFTH OR HYPOGLOSSAL NERVE

Anatomical.—This nerve arises from a nucleus situated in the floor of the 4th ventricle, and its filaments appear on the anterior surface of the medulla near the middle line, whence, uniting into a trunk, they pass through the anterior condyloid foramen.

The muscles chiefly affected in **lesions of the hypoglossal** are those of the tongue.

Lesions of the nucleus of the fibres of the nerve cause loss of power, perhaps at first only showing itself by indistinct pronunciation of certain letters, and later by paralysis so that the tongue cannot be put out or thrust into either cheek. As the muscle atrophies the mucous membrane becomes thrown into folds (Fig. 21).

When the muscles of one half of the organ are weakened the tongue, if protruded, will deviate towards the weakened side.

The nerve supply of the orbicularis oris, though running in the trunk of the facial, appears to come from the hypoglossal nucleus, and hence paralysis of the lips of nuclear origin is liable to accompany that of the tongue, as, for instance, often happens during the course of progressive bulbar paralysis.

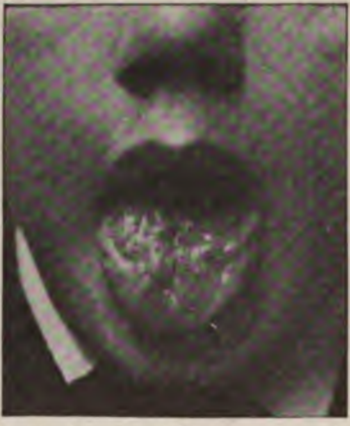

Fig. 21.—Atrophy of the tongue muscles. Note the wrinkled surface.

The hypoglossal nucleus is especially liable to be the seat of degeneration in progressive bulbar paralysis; it may also be affected in syringomyelia of the medullary region and by the pressure of tumours.

At the base of the skull, meningitis and tumours may compress the nerve trunks.

CHAPTER XII

PARALYSES OF THE UPPER LIMB

Anatomical.—The **brachial plexus** is made up of the 5th, 6th, 7th, and 8th cervical roots, together with the 1st dorsal root. The fibres of the roots enter into the composition of different nerve trunks, so that the distribution of the paralysis from a root lesion differs from that due to a lesion of a peripheral nerve.

It is thus necessary, for purposes of localization, to distinguish between paralyses of root-distribution and those of peripheral nerve origin.

On the next page will be found the table compiled by Wilfred Harris* as the result of stimulation of nerves in operation cases, of observations on pathological conditions, and of dissections of the plexus. Harris points out that certain variations may be met with through plexuses being shifted either up or down. If the plexus is a high one (i.e. prefixed) it receives a branch from the 4th cervical nerve with some curtailment of the branches from the lowest roots. If, on the other hand, the plexus is a low one (i.e. postfixed) there will be no branch from the 4th cervical nerve, and the roots at the lower end will be of large size. These differences entail slight variations in the distributions of the roots. The table is from a prefixed type of plexus.

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* "The True Form of the Brachial Plexus and its Motor Distribution," *Journ. of Anat. and Phys.*, July, 1904.

MOTOR ROOT SUPPLY OF MUSCLES OF UPPER EXTREMITY

(*Wilfred Harris*)

PREFIXED TYPE OF PLEXUS

NERVE ROOTS	MUSCLES
4–5th C.	Deltoid.
	Teres minor.
	Supraspinatus.
	Infraspinatus.
5th C.	Rhomboids.
	Subclavius.
	Biceps.
	Brachialis anticus.
	Supinator longus.
	Supinator brevis.
	Pronator radii teres.
	Extensor carpi radialis longior.
	Extensor carpi radialis brevior.
5–6th C.	Clavicular pectoral.
	Subscapularis.
5–6–7th C.	Triceps.
	Serratus magnus.
6–7th C.	Teres major.
	Coraco-brachialis
6–7–8th C.	Pectoralis major (sternal).
	Latissimus.
	Extensor carpi ulnaris.
	Flexor carpi radialis.
7–8th C.	Flexor carpi ulnaris.
	Pectoralis minor.
	Extensor ossis metacarpi pollicis.
	Extensor primi internodii pollicis.
	Extensor communis digitorum.
8th C.–1st D.	Extensor secundi internodii pollicis.
	Extensor indicis.
	Extensor minimi digiti.
	Pronator quadratus.
	Palmaris longus.
	Flexor sublimis digitorum.
	Flexor longus pollicis.
	Flexor profundis digitorum.
1st D.	Thenar muscles.
	Lumbricals.
	Interossei.
	Hypothenar muscles.

It is occasionally possible, in cases of injury of the cord, to analyse further the relative positions of the segments from which the muscles are innervated. I have recorded cases* from which it appears that the extensors and flexors of the wrist are situated at a higher level of the cord than the long flexors and extensors of the fingers and thumb, and, further, that the extensors of the wrist are represented at a higher level in the cord than the flexors of the wrist.

The motor and, of course, in many cases the sensory roots are especially liable to suffer in injuries of the spinal column, e.g. fractures and dislocations, and Farquhar Buzzard has shown that uniradicular palsies apparently depending upon a vascular lesion may sometimes occur. The whole or part of the brachial plexus may be severely injured by dislocations of the head of the humerus, or in elderly people it may be subject to inflammations which appear to be of a rheumatic or gouty nature.

The peripheral nerves may be crushed or severed, they may suffer as part of a more generalized neuritis, or they may be injured by continual pressure as from the use of crutches.

PARALYSES OF THE BRACHIAL PLEXUS

For purposes of classification, the paralyses of the brachial plexus may be grouped in three divisions, though, of course, they are often mixed:

1. Paralyses of the upper type, in which the loss of power corresponds to the distribution of the 5th cervical root.
2. Paralyses of the lower type, in which the weakness chiefly follows the supply of the 1st dorsal root.
3. Paralyses of the intermediate type, in which the fault lies in the 6th, 7th, and 8th cervical roots.

The 5th cervical root, owing to its anatomical position, is the one likely to suffer most when the neck and shoulder are forcibly stretched apart. This sometimes happens to a child as it is being born, with the result that the deltoid, biceps, and supinator longus are paralysed, and occasionally other roots of the plexus are also affected. The condition is often known as **obstetrical paralysis.** The arm is usually kept extended and rotated inwards by the unantagonized action of the triceps and other muscles. Whether recovery takes place or not depends upon the degree of primary injury, though happily in many cases the power of conductivity returns.

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* *Brain*, Part lxxxv., 1899.

The line of **treatment** consists in keeping the paralysed muscles relaxed and preserving as far as possible their nutrition by means of massage and electricity, until the functions of the nerve fibres are restored. If after some months there be no sign of any return of power, and no encouragement be obtained from the electrical reactions, the chances that the injured nerve will recover are very slight. In cases which show no signs of recovering, the possibility of obtaining any improvement from an operative procedure by grafting the injured nerve on to a healthy one must be considered.

The 8th cervical and 1st dorsal roots are sometimes injured, as they leave the cord, by accessory ribs, tumours, or thickening of the meninges (Plate 6). This variety is known as **lower plexus** or **Klumpke's paralysis.** The small muscles of the hand waste, and there is some pain or anæsthesia over the area supplied by the roots. In some cases the oculo-pupillary fibres are implicated, with consequent miosis and narrowing of the palpebral fissure. The segments of the cord supplying the muscles of the hand are especially apt to be affected in progressive muscular atrophy and syringomyelia.

Cervical rib.—The symptoms of a cervical rib are produced by pressure on the roots of the brachial plexus, the 1st dorsal root being the one most commonly affected.

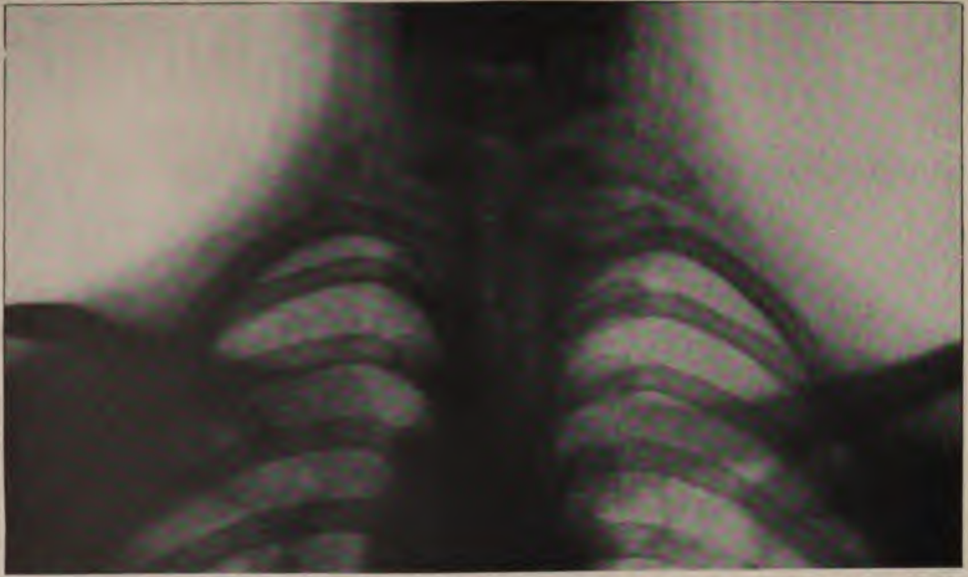

PLATE 6.—Skiagram of a cervical rib which gave rise to symptoms of pressure on the lower roots of the brachial plexus. (The rib was eventually removed by Sir Alfred Pearce Gould.)

Symptoms.—The patient usually complains of weakness of the hand, together with pain, tingling, and numbness chiefly along the ulnar border of the forearm, and on examination some anæsthesia can often be demonstrated over this part, i.e. over the area supplied by the 1st dorsal root. The weakness is accompanied by wasting of the small muscles of the hand, and Kinnier Wilson has pointed out that when the muscles of the thumb are affected the wasting is often limited to the abductor and opponens pollicis, a distribution which is in contrast with that found in progressive muscular atrophy, where all the thumb muscles suffer equally, and which may thus be an additional point of diagnostic importance in differentiating between the two diseases. Signs of pressure on the cervical sympathetic fibres may be met with, as mentioned in the preceding paragraph.

The symptoms caused by cervical ribs usually come on comparatively late in life, and the cause of this lateness of onset has given rise to considerable speculation. It has been attributed to late ossification of the cervical rib, but, as pointed out by Wood Jones, (1) there is no evidence that a cervical rib does ossify late, and (2) this hypothesis must fail when a normal first rib is the offender. The explanation suggested by this observer is that the symptoms are brought about by a change in the relative positions of the different parts of the shoulder girdle consequent on general loss of muscular tone. It is thought that the dropping of the outer end of the clavicle which is apt to occur in such circumstances may lead to tension arising between the lowest cord of the plexus and the cervical rib; and in support of this view it has been shown that symptoms can often be relieved by voluntary elevation of the shoulder girdle. In some cases the symptoms appear to be caused by a ligamentous structure attached to the rib rather than by the rib itself.

Diagnosis.—The diagnosis is not always easy, and it must not be too hastily concluded that the presence of a cervical rib—which, as shown by the X-rays, is not an uncommon occurrence—is necessarily the cause of the symptoms.

Where sensory disturbance alone is complained of, the condition must be differentiated from those symptoms of tingling, pain, and numbness often known as *acroparæsthesia,* which are frequently complained of by women and appear to depend upon vaso-motor derangement quite independently of any gross anatomical lesion.

Assistance in arriving at a conclusion will be derived from observation of the distribution of the symptoms. When a cervical rib is the cause the sensory disturbance will tend to be distributed over a root area, usually that of the 1st dorsal; while in acroparæsthesia of general origin the symptoms are more widely distributed and often occur in both arms and possibly in the legs as well. When wasting of the small muscles of one hand is the prominent symptom the possibility of progressive muscular atrophy or syringomyelia has to be thought of, and here the escape of certain small muscles of the thumb in the cervical rib cases, and the distribution and nature of the sensory changes, are points that will be of assistance.

The most conclusive cases are those in which wasting and sensory changes of gradual onset are associated with the presence of a cervical rib and are confined to the distribution of the 1st dorsal root.

Lesions of peripheral nerves.—Lesions of nerves below the level of the plexus give rise to symptoms corresponding to the peripheral nerve distribution. The accurate investigation of these is of the greatest importance in view of the surgical procedure which is so often required. The signs of injury to a peripheral nerve may be summarized briefly as follows :—

1. *Movement.*—The motor effects of a peripheral nerve injury are those already described under the heading of Lower Motor Neurone Lesions, and can be recognized by (i.) loss of power, (ii.) muscular wasting,

(iii.) loss of reflexes in the affected area, (iv.) changes in the electrical reactions, (v.) late deformities.

2. *Sensation.*—The three peripheral sensory systems for (*a*) light touch, (*b*) pain, and (*c*) deep sensibility, as described at p. 12, must all be carefully tested, and it must be remembered that the areas over which these are affected by no means always correspond with one another. Thus, when a peripheral nerve such as the ulnar is severed, there will be no loss of deep sensibility unless the tendons along which the nerves for deep sensibility run are also severed, and so it is possible for the patient to recognize pressure sensations when he cannot feel those of light touch. Moreover, Head has shown that the proportionate effects on the different peripheral sensory systems vary according as to whether the lesion is nearer the periphery or the cord.

The more distal the peripheral lesion—i.e. the nearer it is to the hand or foot, as in the case of division of the median or ulnar nerves—the greater will be the extent of the loss to light touch as compared with that of pin-prick.

As the position of the lesion gets nearer the central nervous system, as in lesions of the brachial plexus, the loss of the sensation to pin-prick increases in area until it coincides with that of touch, while on the central side of the plexus, as in a lesion of the posterior roots, the area of loss to pin-prick may actually exceed the extent of that for light touch. The proportionate area between loss of pin-prick and of light touch may thus give some indication to the position of the lesion.

3. *Vaso-motor and trophic symptoms.*—Dryness, cyanosis, and coldness of the skin, which also easily ulcerates after any slight injury, are all common results of peripheral nerve injuries. The injured part may become œdematous if kept in a dependent position, and all the vaso-motor symptoms are more pronounced in cold weather.

Treatment.—The general principles underlying the treatment of peripheral nerve injuries consist chiefly in keeping the paralysed muscles in a position of relaxation, maintaining their nutrition by such means as massage, electrical treatment, and exercises, and giving such drugs as may be necessary for the relief of pain. In cases which do not progress satisfactorily the question of *operation* has to be considered, the chief indications for which, as recently laid down by the Medical Research Council,* are (i.) persistence of total loss of conductivity (sensory and motor) after an interval of two months, although proper treatment has been carried out; (ii.) palpable neuromata at the site of injury of a nerve, the function of which is seriously disturbed; (iii.) when recovery has begun but has not progressed at the usual rate or has actually ceased, and still more when function has relapsed; (iv.) persistent, severe, and intractable pain. On the other hand, operations should not be undertaken (*a*) in the presence of an unhealed wound or in recently healed wounds when there is danger of recrudescent sepsis; (*b*) when recovery is progressing (it must not be forgotten that early signs of recovery may be associated with increased discomfort); (*c*) when the locality of the injury is such as to render an operation unusually difficult or the likelihood of successful suture very doubtful.

The **circumflex nerve** is often injured from lesions in the neighbourhood of the shoulder-joint, with resulting paralysis of the deltoid and teres minor together with anæsthesia of an area of skin on the outer aspect of the upper arm over the region of the insertion of the deltoid.

In dislocations of the shoulder-joint the nerve is often stretched, and it is common, even after the dislocation has been reduced quite quickly, to find some weakness of the deltoid, as shown by inability to raise the arm to the horizontal position (Fig. 22). In other cases, especially those of subglenoid dislocation, the whole plexus may be severely contused, and if the head of the bone is not replaced within a reasonable time the results may be disastrous. In such cases the arm is completely paralysed, the muscles waste rapidly, and the skin assumes the characteristic glossy appearance There is usually agonizing pain, with varying degrees of

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* "First Report of the Committee upon Injuries of the Nervous System," 1920. To this the reader is referred for further details concerning injuries to peripheral nerves.

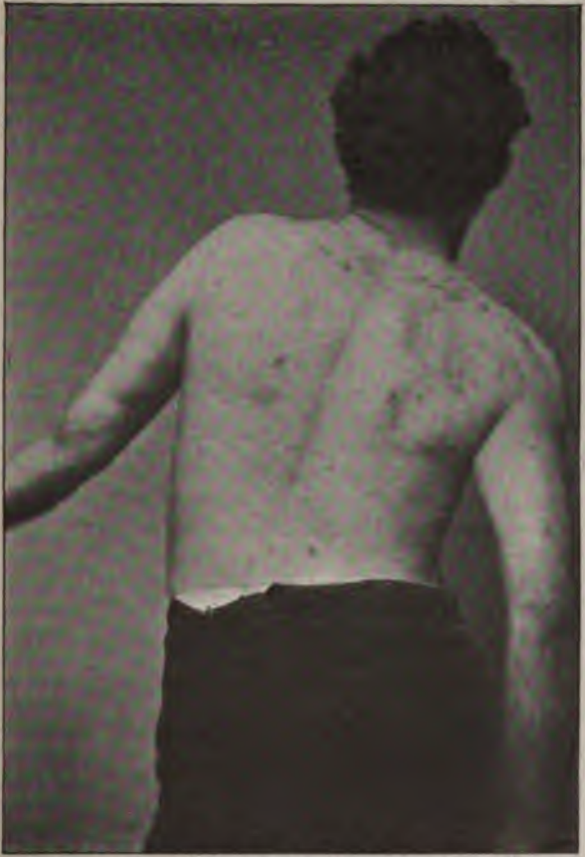

Fig. 22.—Paralysis of the deltoid. Note the position which the patient assumes in endeavouring to raise his arm.

anæsthesia, and often intense hyperæsthesia. Massage and electrical treatment must be given regularly, although at first, on account of the pain, both have frequently to be administered very lightly.

For the pain, local sedatives are often useful, while, internally, phenacetin and similar drugs are beneficial. In many cases morphia is required, if only to produce the sleep that it may be otherwise impossible to obtain. In one case in which the pain was excruciating, some relief was obtained by rapidly alternating applications of heat and cold.

Recovery in these severe cases is very slow, but may in the end be complete, provided methods of treatment are conscientiously carried out by the patient for a long time. One of the most important points in all cases of paralysis of the deltoid is to keep the injured muscle in a state of relaxation, which can be done by fixation of the arm in a position of abduction by means of a suitable splint.

The **pectoralis major,** which obtains its nerve supply from the external and internal anterior thoracic nerves, is an important muscle to examine. It consists of an upper and a lower division, and its action, which is chiefly to adduct the arm, can be shown by holding out the arms horizontally in front of the chest and then pressing them towards the middle line against resistance.

The lower division of this muscle is particularly liable to early atrophy in pseudo-hypertrophic paralysis; it is also sometimes absent at birth (Fig. 23).

The long or posterior thoracic nerve (nerve of Bell).—Injury or disease of this nerve results in paralysis of the serratus magnus. The chief functions of this important muscle are (1) to *fix the scapula,* so that other muscles may take their action from it, and (2) to *rotate the scapula* (in conjunction with the trapezius) when the arm is raised above the shoulder.

When the serratus is paralysed the scapula can no longer be fixed, and when the arm is raised it rotates so that its vertebral border projects and presents the wing-like appearance seen in the illustration (Fig. 24).

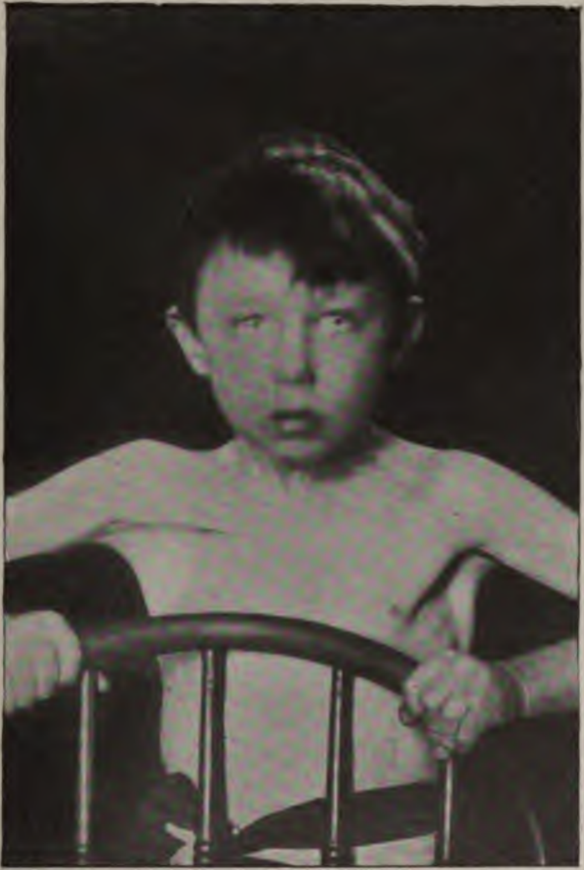

Fig. 23.—Absence (congenital) of the pectoralis major on the right side. (*From a case under the care of Mr. T. H. Kellock.*)

Owing to this want of fixation the power of pushing is also greatly impaired. The diminished power of rotating the scapula is seen on attempting to raise the arm above the level of the shoulder, which movement, in the absence of the serratus magnus, is imperfectly performed by the trapezius. The effects of paralysis of the serratus magnus may sometimes be confused with those due to paralysis of the lower part of the trapezius. The vertebral border of the scapula stands out unduly in both, but, in the absence of serratus magnus paralysis,

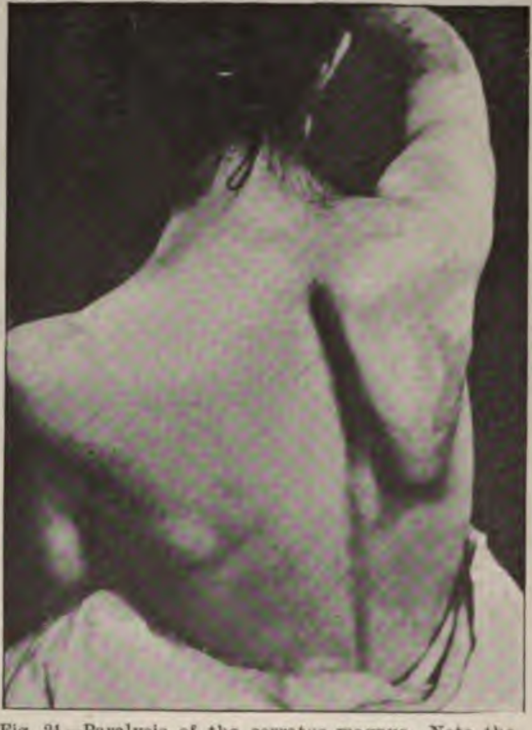

Fig. 24.—Paralysis of the serratus magnus. Note the "winging" of the vertebral border of the scapula when the arm is raised.

the deformity disappears when the scapula is rotated by raising the arm above the head.

There may also be some difficulty in estimating the power of the serratus magnus when the deltoid is paralysed, so that the arm cannot be raised high enough to demonstrate the ability or inability to rotate the scapula. In these circumstances, Duchenne has shown that paralysis of the serratus magnus can be demonstrated by making a forward movement of the shoulder: if the muscle is acting, the lower angle of the scapula is moved forwards and outwards; while if it is paralysed, the spinal border does not move except to become rather more prominent through some rotation on its vertical axis.

Affections of the **long subscapular nerve** result in paralysis of the latissimus dorsi. Contraction of this muscle can be shown by adducting and depressing the arm against resistance.

C. E. Beevor* showed that while only one latissimus dorsi acts in a voluntary adductor movement of the arm, the two muscles act together in such involuntary movements as coughing and sneezing. In cases of hemiplegia it may be possible to show loss of power of the muscle to perform voluntary movements while those for involuntary ones are preserved, just as, in the face of a hemiplegic, the corner of the mouth will not move voluntarily, but will do so in the expression of emotions such as laughing and crying.

Beevor suggested that a knowledge of this double action might at times be useful in differentiating diseases of the brain from those of the cord and peripheral nerves, for whereas in cerebral lesions the involuntary bilateral movements might be preserved, in trouble at the periphery all movements would be lost. He further showed that, though the latissimus has generally been considered to be a muscle of inspiration, its chief action is in reality expiratory in character.

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* *Brit. Med. Journ.*, 1898, ii. 976. As pointed out by Hughlings Jackson, the upper part of the trapezius acts in a similar manner; the highest fibres act unilaterally to elevate the shoulder and bilaterally when called into play to assist in respiration.

The **musculo-cutaneous nerve** arises from the outer cord of the brachial plexus and supplies the coraco-brachialis, the biceps and brachialis anticus. It supplies sensation to the outer side of the forearm, but its distribution is variable, and the resulting changes after injury are usually indefinite in character.

The **musculo-spiral nerve,** the longest offshoot from the brachial plexus, is derived from the posterior cord. At first situated behind the third part of the axillary artery and the commencement of the brachial, it then winds round the back of the humerus in the musculo-spiral groove between the external and internal heads of the triceps, and appears upon the outer aspect of the arm, between the brachialis anticus on the inside and the supinator longus and the extensor carpi radialis longior on the outside; opposite the external condyle of the humerus it divides into the posterior interosseous and the radial nerve. The musculo-spiral nerve supplies the three heads of the triceps, the supinator longus, the extensor carpi radialis longior, the brachialis anticus (in part), the anconeus, the sub-anconeus, and, through its posterior interosseous branch, to the extensor carpi radialis brevior, supinator brevis, extensor communis digitorum, extensor minimi digiti, extensor carpi ulnaris, extensor indicis, and the extensors of the thumb.

The musculo-spiral nerve may be injured by penetrating wounds from bullets or other weapons. It is also apt to be compressed by callus resulting from a fracture of the shaft of the humerus, especially in the neighbourhood of the musculo-spiral groove. On the inner side of the arm the nerve may be injured by the pressure of crutches or by the pressure induced by the arm hanging over a chair or other hard, resisting object, especially if the patient is under the influence of alcohol or other narcotic. The wrist-drop which sometimes occurs after the arm has been lain on during heavy sleep is generally attributed to injury of the nerve, but it seems probable, as suggested by Sir Robert Jones, that in many such cases the paralysis may be due to overstretching of muscles rather than to injury of the nerve.

The fibres of the nerve, with the exception of those going to the supinator longus, are especially liable to be affected in chronic lead-poisoning. (*See* p. 156.)

The chief results of injury to the musculo-spiral nerve are loss of power in the triceps and supinator longus, together with weakness of the extensor muscles of the forearm, giving rise to wrist-drop and weakness of extension of the fingers at the metacarpo-phalangeal joints, the signs differing according to the level of the lesion.

The **posterior interosseous nerve** winds round the neck of the radius in the substance of the supinator brevis (which it supplies) to reach the back of the forearm, and then runs down between the superficial and deep extensors on the back of the forearm; these it innervates, as described on p. 128.

The **radial nerve** is purely sensory, and is distributed to the dorsal aspect of the thumb as far as the bed of the nail, and to a varying extent of surface on the back of the index, middle, and ring fingers. The overlapping of this nerve with the external cutaneous branch of the musculo-spiral is, however, such that no definite anæsthesia usually follows a lesion of the radial unless the external cutaneous branch is at the same time affected.

The **prognosis** depends on the nature and degree of injury. Paralysis from temporary pressure on the musculo-spiral trunk is usually favourable. A few days or a week generally suffices to restore power, but sometimes a much longer period is required, and recovery appears to be a distinctly slower process in elderly people. During treatment care must be taken to keep the paralysed muscles of the forearm relaxed by extension of the wrist and fingers at the metacarpo-phalangeal joints by means of a splint for the wrist and elastic extension for the fingers and thumb. The outlook after suture of the musculo-spiral nerve is good.

The **ulnar nerve,** arising from the inner cord of the brachial plexus, runs along the inner side of the arm, and enters the forearm by passing between the internal condyle of the humerus and the olecranon. In the forearm it runs down upon the surface of the flexor profundus digitorum between the flexor carpi ulnaris on the inside and the flexor sublimis digitorum on the radial side; in the lower two-thirds of the forearm it is closely associated with the ulnar artery, lying upon the ulnar side of the vessel. It passes in front of the anterior annular ligament and divides into a superficial and a deep portion. In the forearm it innervates the flexor carpi ulnaris and the ulnar half of the flexor profundus digitorum, and it often gives off its dorsal cutaneous branch as high as the middle third of the forearm, which supplies the ulnar portion of the dorsum of the hand, and the dorsal aspect of the inner one and a half digits as far as the middle of the phalanges.

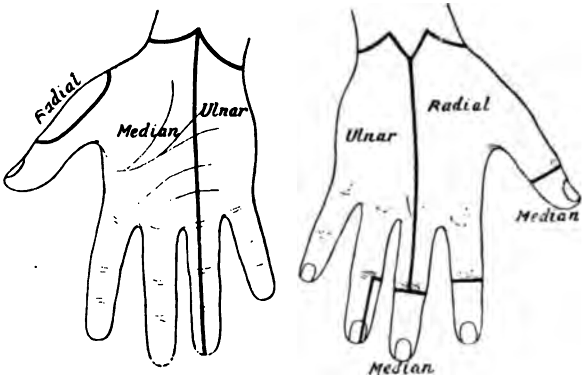

Fig. 25.—Distribution of the sensory nerves in the hand. (*After Bernhardt.*)

The nerve also supplies most of the intrinsic muscles of the palm, namely, the seven interossei, the two inner lumbricals, the three muscles of the hypothenar eminence, the two adductors of the thumb, and the deep head of the flexor brevis pollicis; it supplies, too, the skin over the palmar aspect of the inner one and a half digits, sending branches on to the dorsum as far up as the middle and second phalanges. (Fig. 25.)

The results of ulnar paralysis (Fig. 26) are loss of power, with wasting of the muscles mentioned above. Weakness of the interossei shows itself by inability to separate the fingers in the horizontal plane, and the wasting becomes obvious from the hollows between the metacarpal bones, especially the concavity of the radial side of the first metacarpal, due to loss of substance of the first dorsal interosseous (the abductor indicis). The slight degree of abduction of the index and little fingers which can still be performed by the extensors and lumbricals must not be mistaken for the action of the interossei.

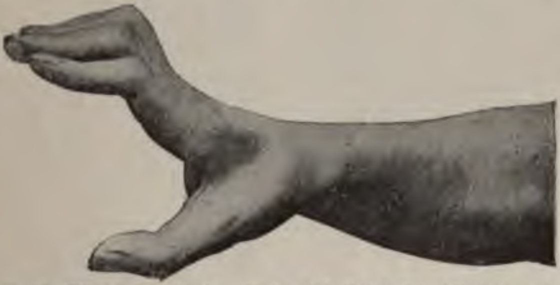

Fig. 26.—Ulnar paralysis. Note the "claw-hand" shape, and the hollow caused by the wasting of the first interosseous (abductor indicis).

Loss of adduction of the thumb, i.e. the movement of the thumb which brings its ulnar border alongside the metacarpal bone of the first finger, can be shown by asking the patient to press something between the ulnar border of the thumb and the first finger. Weakness of this movement indicates loss of power of the adductor and inner head of the flexor brevis pollicis, and a hollow over the site of these muscles is often visible. Abduction of the index and little fingers can be performed to a limited extent by their extensor tendons, and the contraction of the first lumbrical in the first space may be mistaken for that of the abductor indicis. After paralysis has lasted for some time the hand tends to assume the claw position (*main-en-griffe*), which must be distinguished from Volkmann's contracture (*see* p. 136).

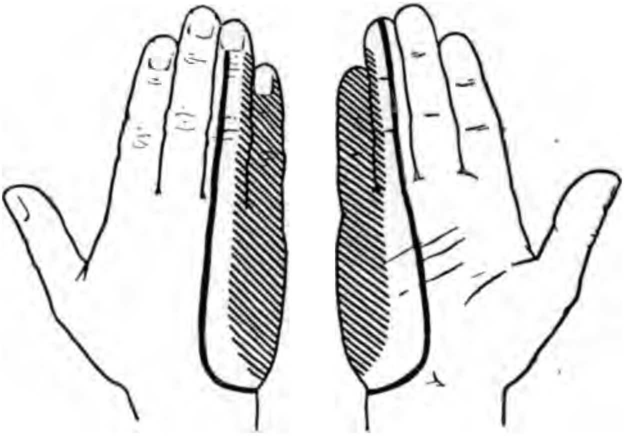

Fig. 27.—Loss of sensibility after complete division of the ulnar nerve. (*Sherren, in Choyce's System of Surgery.*)*

The extent to which anæsthesia is produced by injuries of the ulnar nerve differs according as the lesion is situated above or below the origin of the dorsal cutaneous branch. When the injury is above the origin of the dorsal branch the loss of sensation is distributed over the ulnar side of the hand, including the whole of the little and half the ring fingers (Fig. 27). When, however, the nerve is injured just above the wrist—the most frequent site of injury—the dorsal branch escapes, and the extent of the anæsthesia will consequently be much less, the patient retaining sensation over the proximal parts of the dorsal aspects of

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* In this diagram, and in Figs. 28 and 29, the area included within the firm line is that of insensibility to light touch; the shaded area, that of insensibility to prick as well as to light touch.

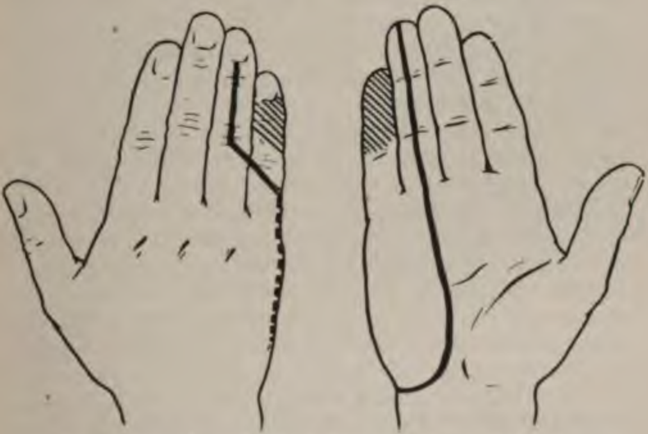

Fig. 28.—Loss of sensibility following division of the ulnar nerve below its dorsal branch. (*Sherren, in Choyce's System of Surgery.*)

See footnote on p. 132.

the ring and little fingers while the peripheral portions of these digits are anæsthetic (Fig. 28). In both cases the area of anæsthesia, as seen in the figures, is more extensive to light touch than to painful stimuli, owing to the overlapping which takes place in the fibres for the latter.

The impulses for deep sensibility are conveyed along the nerves of muscles and tendons, and unless these latter are injured the patient will be able to recognize the sensations of passive positions, deep pressure, and the vibrations of a tuning-fork.

The **median nerve,** being less exposed, is less liable to injury than the ulnar, but may be damaged in fractures and dislocations of the humerus. It is most frequently injured above the wrist in such accidents as the bursting of bottles or thrusting the arm through a window.

The median nerve arises by two heads, one from the outer and the other from the inner cord of the brachial plexus. The nerve is first situated on the outer side of the axillary and the brachial arteries, but in the middle of the arm it crosses the latter vessel from without inwards, and runs down on the inner side as far as the elbow. It passes between the two heads of the pronator radii teres, being separated from the ulnar artery by the deep head of the latter muscle, and then continues straight down the forearm between the flexor sublimis and flexor profundus digitorum muscles; just above the anterior annular ligament it lies exactly posterior to the palmaris longus tendon, if this is present. It passes behind the anterior annular ligament enveloped by the synovial sheath which is wrapped round the tendons of the flexors of the fingers, and, reaching the palm, divides to supply the muscles of the thenar eminence, viz. the abductor pollicis, the opponens pollicis, and the superficial head of the flexor brevis pollicis. It innervates the two outer lumbricals, and supplies the skin over the palmar aspect of the radial side of the hand, over the front of the thumb, the index, middle, and half the ring fingers, and over varying portions of the dorsum of the same.

In the forearm the median supplies all the muscles arising from the internal condyle, except the flexor carpi ulnaris; through its anterior interosseous branch it innervates the deep muscles of the forearm, viz. the flexor longus pollicis, the pronator quadratus, and the outer half of the flexor profundus digitorum (the inner half of the latter muscle deriving its nerve supply from the ulnar nerve).

In cases of lesion of the median nerve there will be loss of power in the muscles supplied by the nerve, and anæsthesia over the area of skin which it innervates; but the action of some of the muscles to which the median is distributed may be closely simulated by others drawing their nerve supply from a different source. Flexion of the wrist, for instance, can be performed by ulnar muscles, and also to some extent by the extensor ossis metacarpi pollicis.

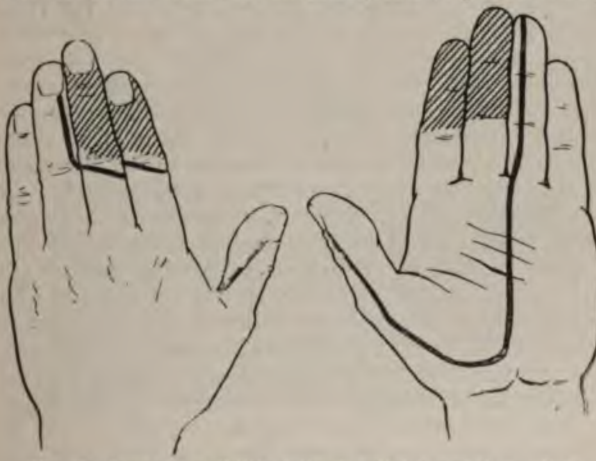

Fig. 29.—Loss of sensibility following division of the median nerve. (*Sherren, in Choyce's System of Surgery.*)

See footnote on p. 132.

Action of the abductor brevis pollicis can be closely simulated by that of the extensor ossis metacarpi pollicis, but, as Beevor pointed out, true abduction of the thumb takes place in a plane at right angles to that of the palm, and abduction by means of the extensor ossis metacarpi pollicis is always complicated by extension of the thumb at the same time.

Sensation to light touch is abolished over the area of skin supplied by the nerve, but the extent to which sensation to pin-prick is lost varies, and is usually much less than that of touch. (Fig. 29.) Presence or loss of deep sensibility will depend upon the integrity or otherwise of the tendons along which the nerves for this form of sensation run.

Volkmann's Contracture, or Ischæmic Paralysis

This form of paralysis was first described by Volkmann in 1875, and is characterized by a contraction of the forearm muscles, which causes contraction of the fingers and sometimes also of the wrist.

Etiology.—The affection usually arises during treatment for fracture in the region of the elbow-joint, especially in connection with fractures of the lower end of the humerus, or a separation of the lower epiphysis of that bone. *Undue pressure by splints and tight bandaging* appears to play the most important part in the production of the contracture, as is evidenced by the fact that in a large number of the cases the splints exercised sufficient pressure to cause sloughing. It is not so much that the splint is applied too tightly at first, as that no allowance is made for the swelling that is likely to ensue in the next few days.

Prolonged obstruction to the circulation by an Esmarch's bandage, injury to the brachial artery, rupture or severe contusion of the flexors, and prolonged suppuration in the muscles have also been assigned as causes of the contracture.

The above condition is most frequently met with in children.

Symptoms.—The chief characteristics are *hardness of the muscles*, followed by *contraction of the flexors of the forearm*, causing flexion of the fingers, and sometimes of the wrist; and in severe cases the forearm is fully pronated and fixed in this position. When the wrist is flexed it may be possible to uncoil the fingers to a varying degree, but as soon as the wrist is extended the pull of the tightened flexors is felt, and the hand again assumes a claw-like aspect, the metacarpo-phalangeal joints being in a condition of hyperextension and the middle and terminal phalanges clenched in extreme flexion. (Figs. 30, 31.)

Pathology.—The exact nature of the condition is not quite clear, and the term "ischæmic" has been applied on the supposition that the pressure of the splints induces the changes by arresting the circulation in the muscles. The rapidity with which the symptoms show themselves, together with the fact that in some cases there are no alterations in the electrical reactions and no anæsthesia, supports the hypothesis that the lesion is primarily one of muscular rather than of nervous origin. The pale, firm, fibroid condition of the muscles is also in favour of this view, and the very early onset of the contracture in some cases, supervening even in a few hours, suggests the presence of some coagulation process or local "rigor mortis" of the compressed fibres as the pathological condition rather than a myositis, though this very early onset may possibly be due to nerve spasm. On the whole, an inflammatory condition of the muscular fibres seems the most likely cause of the contracture.

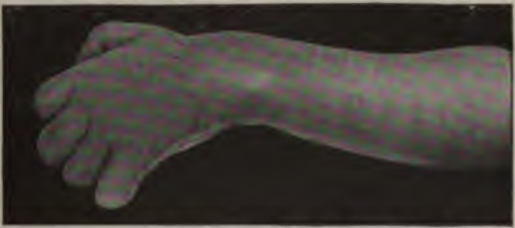

Fig. 30.—Position of hand in an early stage of ischæmic paralysis.

In some cases electrical changes and anæsthesia have been observed, but this fact does not necessarily invalidate the hypothesis that the disease is of muscular origin, for it is quite possible for injuries to the nerves to coexist with Volkmann's contracture, and sometimes the median and ulnar nerves have been found embedded in scar tissue, but otherwise healthy in appearance.

Diagnosis.—The diagnosis is based upon the history, the character of the deformity, the absence of electrical changes, the frequent absence of anæsthesia, and the simultaneous onset of paralysis and contracture.

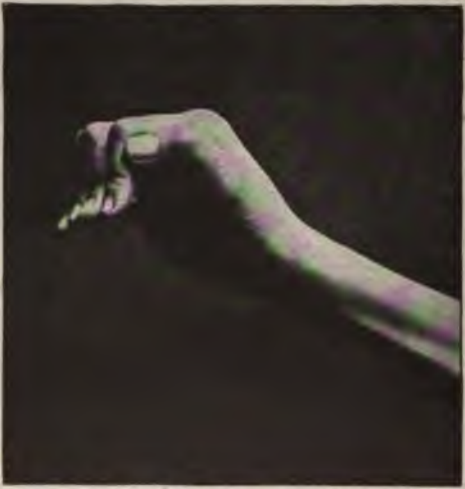

Fig. 31.—Position of fingers caused by contraction of the flexor tendons in ischæmic paralysis.

Ulnar paralysis, in its later stages, to a certain extent resembles Volkmann's contracture, but in the former there is no flexion of the wrist, while the small muscles of the hand (except those supplied by the median) are wasted and show the reaction of degeneration. Further, there may be anæsthesia or paræsthesia over the area of skin supplied by the ulnar nerve.

Late musculo-spiral paralysis.—In this condition the extensors of the wrist and fingers are paralysed and give the reaction of degeneration. The supinator longus and triceps may share in the paralysis, and the terminal phalanges can still be extended by the interossei and lumbricales.

Infantile paralysis (acute poliomyelitis) is distinguished from Volkmann's contracture by the history, the presence of the reaction of degeneration, and the fact that contracture follows the paralysis at a later date.

Treatment.—Massage, passive movements, and electricity may lead to much improvement in mild cases, but they must be commenced early and practised regularly. Even in the severe cases, although these measures fail to cure the deformity, they are nevertheless of value in maintaining the nutrition of the muscles, and should be given a fair trial for three or four months before any treatment by operation is considered.

Prognosis.—Some mild cases may recover by the systematic employment of active and passive movements, massage, and electricity, but in many cases operation is necessary to permit of a more extensive range of movement for the diseased muscles, and to correct the deformity, before the former method of treatment can be of any permanent value.

and paræsthesia of various kinds are apt to occur in the distribution of this nerve.

The **anterior crural nerve** springs from the 2nd, 3rd, and 4th lumbar roots, which unite to form one trunk between the psoas and iliacus muscles. Besides sending branches to the iliacus, pectineus, and sartorius, it also supplies the quadriceps extensor, which is composed of the rectus, vasti, and crureus. The middle and internal cutaneous nerves, which supply the skin over the lower two-thirds of the anterior and inner surfaces of the thigh, and the long saphenous nerve, which supplies sensation over the anterior and inner part of the leg, all arise from the anterior crural.

The chief motor result of disease of the anterior crural nerve is loss of power to extend the knee, from paralysis of the quadriceps extensor. The knee-jerk is diminished or lost. Flexion of the hip is weakened through paralysis of the iliacus, but it can still be carried out by the psoas, which is supplied by special branches from the 2nd and 3rd lumbar nerves. Sensation may be impaired over those parts of the leg and thigh which the nerve supplies.

The **superior gluteal nerve** arises from the lumbosacral cord and the 1st sacral nerve. It is responsible for the nerve supply of the gluteus medius and minimus, and of the tensor vaginæ femoris.

The **inferior gluteal nerve** supplies the gluteus maximus. The gluteus maximus is one of the principal extensors of the thigh, but is also an abductor and outward rotator. Its action can be tested by asking the patient to lie flat on his face and then to raise his thigh. The gluteus maximus is also of importance from the frequency with which it is enlarged in pseudo-hypertrophic paralysis. The gluteus medius and minimus are both abductors, but they also to some extent flex and rotate the thigh inwards.

The **small sciatic nerve** most usually arises from the 1st, 2nd, and 3rd sacral roots. After running down the back of the thigh and giving off branches which supply the skin over the back of the thigh and popliteal space, it pierces the deep fascia of the leg below the knee and supplies the skin over the upper part of the calf.

GREAT SCIATIC NERVE AND ITS BRANCHES

The great sciatic nerve arises from the sacral plexus at the lower border of the great sacro-sciatic foramen. It runs down the back of the thigh and terminates by dividing into the external and internal popliteal nerves.

As the great sciatic nerve passes down the thigh, branches leave it to supply the biceps femoris, semimembranosus, semitendinosus, and a portion of the adductor magnus, which may all suffer in severe cases of sciatica.

Paralysis of the sciatic nerve may be caused by injuries of different kinds, and the nerve trunk may be divided by stabs or bullet wounds. In this connection it is important to remember that the two main divisions of the nerve, i.e. the internal and external popliteal branches, although contained in the one sheath, remain separate from one another up to their origin from the sacral plexus. It is therefore possible in injuries of the main trunk for one division to be damaged while the other escapes. Complete division of the sciatic nerve is followed by loss of power in all the muscles which it supplies, and loss of sensation over a considerable portion of the foot and leg, as shown in Fig. 32.

At the top of the popliteal space the main trunk divides into external and internal popliteal nerves.

The **external popliteal nerve** (peroneal nerve) descends obliquely along the outer side of the popliteal space, running close to the biceps tendon. It then winds round the neck of the fibula (in which position it is very liable to injury), and, passing between the latter and the peroneus longus, divides into the anterior tibial and the musculo-cutaneous nerves.

The external popliteal nerve may be injured in punctured wounds of the upper part of the popliteal space or at the neck of the fibula, and also when the biceps tendon is divided subcutaneously. The peronei, tibialis anticus, and extensors of the toes will be paralysed, and there will be loss of sensation over the area of skin supplied by its two divisions, the anterior tibial and musculo-cutaneous nerves.

The **anterior tibial nerve,** commencing between the fibula and the peroneus longus, inclines obliquely beneath the extensor longus digitorum to reach the anterior surface of the interosseous membrane. It runs down the front of the limb till it reaches the level of the ankle-joint, where it divides into internal and external branches, the former supplying the integument on the neighbouring sides of the great toe and the second toe on their dorsal aspect, the latter being distributed partly as motor branch to the extensor brevis digitorum, and partly as sensory twigs to the dorsal interossei. In the leg the anterior tibial supplies the tibialis anticus, extensor proprius hallucis, extensor longus digitorum, and peroneus tertius.

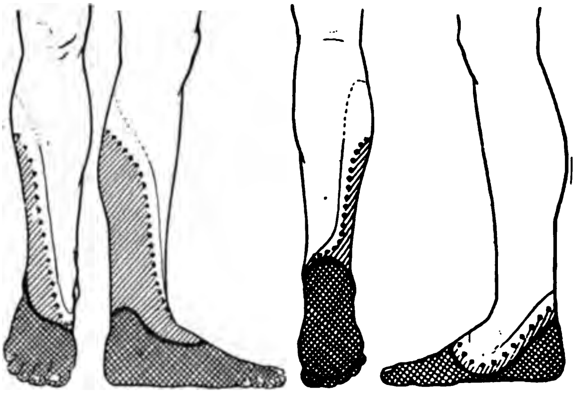

Fig. 32.—Effects of complete division of the great sciatic nerve. The area of loss of sensibility to light touch is bounded by a thin line; that to prick, by crosses; that to all forms of sensibility, including deep touch, by a thick line. (*Sherren, in Choyce's System of Surgery.*)

Paralysis of the anterior tibial nerve leads to *foot-drop*. The foot cannot be flexed on the leg, and the difficulty in walking, consequent on the toes catching in the ground, gives rise to a "high-stepping" gait.

The **musculo-cutaneous nerve,** lying in a sheath in the intermuscular septum between the peronei externally and the extensor longus digitorum internally, runs down in front of the fibula to the lower third of the leg, where it pierces the deep fascia in two branches, internal and external. The main trunk innervates the peroneus longus and brevis, and of the two terminals the internal branch comes down over the anterior annular ligament and supplies the lower third of the leg, the skin over the dorsum of the foot, the inner side of the big toe, and the adjacent side of the second and third toes. The external branch supplies the adjacent sides of the third and fourth, and fourth and fifth toes, communicating with the external saphenous nerve. The action of the peroneus longus and brevis is to evert the foot and also to aid in extending the ankle-joint.

When these muscles are paralysed the power of eversion is lost, and the foot is turned inwards by the unantagonized action of the tibialis anticus and posticus.

The **internal popliteal** passes onwards through the popliteal space, and at the lower border of the popliteus becomes continuous with the **posterior tibial nerve.** The latter runs down the back of the leg and terminates in the **internal** and **external plantar nerves.** The internal popliteal and posterior tibial nerves supply the gastrocnemius, soleus, plantaris, popliteus, tibialis posticus, flexor longus digitorum, and flexor longus hallucis.

In paralysis of these muscles there is loss of power to extend the foot on the ankle-joint and to flex the toes. In long-standing cases the unantagonized action of the flexors produces a condition of talipes calcaneus, in which only the heel can be placed on the ground.

The *internal* plantar nerve is mainly sensory, supplying the palmar surface of the inner three and a half digits, but also innervating four muscles, i.e. the abductor hallucis, flexor brevis hallucis, flexor brevis digitorum, and the 1st lumbrical. The *external* plantar supplies the remaining sensory area and all the other muscles of the foot. As the digital branches from the plantar nerves run forwards to supply the skin covering the toes they pass between the heads of the metatarsal bones, and, as a result of the crowding together of the bones by pressure of tight boots, the nerves going to supply adjacent sides of the fourth and fifth toes may be compressed. Pain may not only be felt at the actual site of pressure, but may radiate up nearly as high as the knee.

SCIATICA

Sciatica is one of the most common of the nervous diseases.

The **causes** may be divided into (1) *extrinsic*, such as pressure from pelvic tumours and spicules of bone, and irritation from rheumatoid changes in the hip-joint, and (2) *intrinsic*, depending upon inflammatory processes in the nerve trunk. These are often brought about by rheumatic and gouty conditions, and in some cases lead may be a factor. Injury, strain, damp, and cold also appear to be causes.

Pathology.—An interstitial inflammation of the nerve sheath with subsequent injury to the nerve fibres is the most common lesion.

Symptoms.—The main symptoms are pain and tenderness. The pain is distributed in the course of the nerve in the thigh, and is often continued down the branches of the nerve beyond. There may be hyperæsthesia, and in a few cases some degree of anæsthesia. In severe cases, tenderness may be elicited by deep pressure anywhere along the line of the nerve, and it is nearly always present at certain points, e.g. the sciatic notch, the popliteal space, and the outer side of the fibula, at which places the nerve is more readily reached.

The pain is often very intense and may prevent the patient from sleeping. The position of greatest ease is obtained by flexing the leg at the knee and hip, and any attempt to extend the leg at once stretches the nerve and causes pain. The position in which the leg is kept causes the patient to stand and walk in such a manner that the weight is chiefly thrown on to the opposite limb, and in time this attitude may cause some lateral curvature of the spine.

In severe and long-standing cases there may be atrophy of muscles, but in the recent and slighter cases there is often no definite loss of power. Occasionally œdema of the leg and herpes are present. The tendo-Achillis jerk is sometimes lost on the affected side; the knee-jerk generally remains, and may be somewhat exaggerated. Lumbago is often associated with sciatica.

Diagnosis.—The diagnosis of sciatica is important, since it is necessary to be careful not to overlook possible causes of pressure. The pelvis must be examined for signs of tumour, and the spine for signs of tuberculosis, with which a psoas abscess may be associated. Account must also be taken of any injury or disease of the pelvic bones and hip-joint (e.g. rheumatoid arthritis), the effects of which may have extended to the sciatic nerve.

The lightning pains of tabes sometimes settle in the course of the sciatic nerve for days at a time. Unless this possibility is remembered, their nature is likely to be overlooked. Suspicion should always be aroused if the "sciatica" is bilateral.

Treatment.—At the outset of an acute attack the patient should be kept in bed and given a purge. To relieve the pain, all the ordinary forms of counter-irritation can be tried, and of these heat often proves the most beneficial, while phenacetin, aspirin, and drugs of similar nature may be given by the mouth. In severe cases, drugs may be administered locally by cataphoresis. Where the pain is very severe, and not relieved by other measures, morphia may be required. Applications of the galvanic current may also help to relieve the pain.

As soon as the more acute symptoms have subsided, massage and electricity may often be prescribed with success. Massage must generally be light at first, though occasionally patients obtain considerable after-benefit from kneading in the acute stages, which at the time gives rise to a good deal of pain. In the more chronic cases, applications of the high-frequency current are often useful.

If, after the acute stage of the disease has passed off, no improvement takes place, the question of nerve injections may be considered. In the case of the sciatic nerve, normal saline in combination with some novocain is generally used, and care must be taken not to inject alcohol or any other substance that will lead to paralysis. Wilfred Harris recommends that the nerve be first injected near the great sciatic notch with 2 c.c. of 2-per-cent. novocain solution, and then at the same point, immediately afterwards, with 70 to 100 c.c. of 0·9-per-cent. saline.

Prognosis varies. Some patients recover rapidly, while in others the disease becomes chronic and lasts for weeks or months. There is a considerable tendency to recurrence.

LOCAL PARALYSIS FOLLOWING SUPERFICIAL INJURIES NOT INVOLVING NERVE TRUNKS.

Walshe* has recently reviewed an interesting group of cases characterized by signs of a local neuritis following a trivial superficial injury to the distal extremity of a limb. He thinks such cases for the most part approximate to the type known as ascending neuritis, rather than to reflex effects such as may follow an acute arthritis, and also considers that a small proportion of the cases described by Babinski as "reflex nervous disorders"† may belong to the same class.

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* *Brain*, Vol. xlii., Pt. iv., 1919.

† During the war Babinski collected a number of cases which he grouped under the heading of "Troubles Nerveux d'Origine Reflex," but doubt has been expressed whether the group was not made up of various peculiar abnormalities for which no common origin could be assigned.

The Cauda Equina

In lesions of the cauda equina the paralysis is of the lower neurone type, characterized by loss of power, wasting, and changes in the electrical reactions of the muscles affected. Control over the sphincters is generally lost, as also is sexual power. Loss of sensation is extensive, and its usual distribution over the buttocks in a "saddle-shaped" area is especially characteristic.

CHAPTER XIV

MULTIPLE NEURITIS

In multiple or polyneuritis there is a generalized inflammation of some or all of the fibres forming nerve trunks. The nerves of the limbs are mainly affected. The symptoms are generally symmetrical, but vary according to the acuteness of the onset and the selective action which different poisons tend to exert.

Selective influence of poisons.—Many poisons which attack the nervous system have an apparent affinity for certain neurones before they produce more generalized symptoms, and a knowledge of their special tendencies is often an aid to diagnosis.

Thus, lead shows a special tendency to affect the nerves of the forearms, alcohol those of the legs, and diphtheria those of the soft palate and eyes.

Etiology.—Of the poisons taken directly into the system, those which most frequently produce neuritis are *alcohol*, *lead*, and *arsenic*, and, less frequently, copper and mercury. Toxins which are the direct outcome of bacterial or protozoal infection are also causes, e.g. those associated with *diphtheria*, *typhoid*, *malaria*, *pneumonia*, *tubercle*, and other infectious diseases. Of these the diphtheria toxin is by far the most important, and, indeed, peripheral neuritis following the others is distinctly uncommon. The toxin of acute poliomyelitis also sometimes attacks the peripheral nerves.

The toxins associated with *leprosy* and *beriberi* are potent causes in countries where these diseases are prevalent.

Other poisons, probably of chemical rather than of bacterial origin, which may cause neuritis are those associated with *gout*, *rheumatism*, *anæmia*, and *diabetes*. Neuritis may also follow injuries and the general enfeeblement attending wasting diseases and senile degeneration.

Pathology.—The changes found in the nerves in multiple neuritis are those of a parenchymatous inflammation and degeneration, to which may be added more diffuse inflammatory changes in the interstitial tissue and nerve sheaths, according to the nature and virulence of the poison. As a general rule the changes are most advanced in the terminal filaments.

In many instances some changes have been observed in the motor cells of the cord also, thus showing that the poison has a tendency to attack the whole of the lower motor neurone.

General symptomatology. — An ordinary "mixed" nerve contains motor and sensory fibres, and possibly some fibres which exercise a trophic influence, although the independent existence of these is doubtful. A peripheral nerve may thus be divided into its various component fibres, like the columns of the cord, and, as in the latter, all the parts need not be affected equally.

When the functions of all the fibres are impaired or destroyed, there are *loss of power, loss of sensation*, and *loss of reflexes*, with perhaps some *trophic* changes in the skin, which last are not, however, of so acute a nature as those found in myelitis.

The *loss of power* is mainly in the limbs, and may be confined to the legs or to the arms, or may be present in both. The weakness is usually most marked in the extensor muscles below the knees and elbows, giving rise to "dropped foot" and "dropped wrist." The trunk muscles and those supplied by the cranial nerves are more seldom affected, but the pneumogastric and phrenic nerves are especially liable to involvement in postdiphtheritic neuritis.

The signs are those of a lower neurone lesion, viz. wasting and alterations in the electrical reactions of muscles, loss of reflexes, and a tendency for the limbs to assume abnormal positions. The rapidity with which power is lost varies greatly, and in some of the acute toxæmic cases the limbs may become powerless in even a few hours. The knee-jerks are generally lost early, but occasionally they are temporarily exaggerated during the period of nerve irritation.

Sensory symptoms are often among the earliest to appear. Apart from complete anæsthesia, irritation of the sensory fibres can cause every variety of disturbance of sensation, including pain, tingling, numbness, and hyperæsthesia of different intensities. When definite anæsthesia develops, it is usually symmetrical in the limbs, and corresponds more or less to the areas over which gloves and stockings are usually worn, and hence it has received the name of "glove-and-stocking anæsthesia." The anæsthesia does not extend to the trunk. The muscles of the legs are often very tender, especially in the alcoholic cases, and in some instances loss of muscle sense, with consequent incoördination, is one of the most prominent symptoms. The intensity and rapidity of the onset of sensory symptoms are very variable; in most of the chronic cases the subjective sensations of tingling, numbness, and pins-and-needles come on gradually and are the first symptoms to which attention is drawn. The extent to which the different forms of sensation are impaired is also very variable: in some patients all forms are affected; in others the loss of muscle sense may be disproportionately great, with a resulting ataxy that may be mistaken for that of tabes. In some of the more acute varieties of toxic origin sensations may be but little affected.

Vaso-motor and **trophic symptoms** are not, as a rule, prominent. Œdema of the legs may be occasionally observed. The "trophic" changes show themselves chiefly by impaired nutrition of the skin, which becomes smooth and glossy; of the nails, which become brittle and lined; and of the smaller joints, which are apt to become stiff and to contract adhesions. Acute bedsores do not occur, and control over the sphincters is retained.

ALCOHOLIC NEURITIS

Etiology.—Alcoholic neuritis occurs from excessive drinking over a long period of time. Spirits appear to be the more potent cause, but many cases arise from drinking beer. Both men and women are affected.

Symptoms.—The *loss of power* nearly always begins in the feet and legs, and is most marked below the knees. The feet drop into a line with the legs (*foot-drop*), and if the patient is able to walk the gait is *high-stepping* in order to clear the toes from the ground. The arms are sometimes affected, and palsies of cranial nerves have occasionally been observed. The *wasting* of the paralysed limbs is generally very pronounced, and the *electrical reactions* are those of degeneration. The *knee-jerks*, *tendo-Achillis jerks*, and *plantar* reflexes are usually lost.

The **sensory symptoms** are prominent. Thus, pain is often excruciating, and the muscles of the calves may be very tender on pressure, although the latter sign is by no means always present. Numbness and tingling are common, as also is hyperæsthesia. The anæsthesia takes the characteristic "stocking-and-glove" distribution already described. The muscle-sense is apt to be greatly diminished, giving rise to ataxy resembling that of tabes.

The *vision* occasionally suffers; there may be amblyopia or impairment of colour vision, and nystagmus and optic neuritis have both been known to occur, though very rarely.

Mental symptoms.—Emotional tendencies, untruthfulness, indecision, mental confusion, with loss of memory for recent events and loss of ideas of space and time—symptoms which are grouped under the designation of

Korsakow's psychosis—are generally met with in some degree, while in the more severe cases hallucinations, delirium tremens, or insanity may be present. These mental changes are sometimes associated with loss of control over the sphincters — a symptom which is not present in uncomplicated cases of multiple neuritis.

Pathology.—The chief changes are parenchymatous inflammation and degeneration of nerve fibres, the terminal twigs suffering most severely. The cells of the cord and cortex may also be altered, and it is to changes in the latter that the cerebral symptoms are due.

ARSENICAL NEURITIS

Etiology.—Arsenical poisoning has occurred from the inhalation of particles arising from various decorative articles into the composition of which arsenic has entered, as green wall-papers and carpets. Arsenic may also accidentally contaminate articles of diet in sufficient quantities to cause symptoms, as occurred in an epidemic among beer-drinkers due to the presence of arsenic in glucose. Neuritis may also be caused by long-continued use of arsenic as a medicine.

Symptoms.—There is a close similarity between the symptoms of arsenical neuritis and those of alcoholic neuritis, to which the reader is referred.

The legs are generally affected first (Fig. 33), and pain, tingling, and numbness are accompanied by weakness of the extensors and by ataxy. Later on, the paralysis may appear in the arms.

Arsenical neuritis is accompanied by pigmentation of the skin, which is a symptom of diagnostic importance. The skin assumes a deep-brownish colour, which is most apparent on the trunk and extensor surfaces of the limbs. Herpes, keratosis of the palms of the hands and soles of the feet, erythema, erythromelalgia, changes in the structure of the nails, and œdema of the extremities may all be present. Recovery generally takes place very slowly.

LEAD NEURITIS

Etiology.—This form of neuritis is chiefly found in painters, who inhale the poison, and, unless they exercise great care, also contaminate their food with particles that have clung round the finger-nails. Workers with metallic lead, such as type-setters, are also liable to the disease. Another source of poisoning is the contamination of water or other drink with lead.

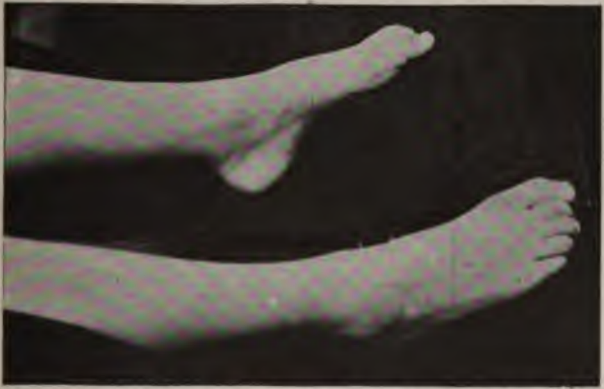

Fig. 33.—"Foot-drop" in arsenical neuritis. *(By courtesy of Dr. George Ogilvie.)*

Symptoms.—Neuritis does not usually occur until the patient has been exposed to the influence of lead for a long time, and it is generally preceded by other indications of lead poisoning, such as colic, dyspepsia, constipation, and anæmia.

The characteristic blue line on the gums can also often be observed, and analysis of the urine and fæces may afford confirmatory evidence of the presence of lead in the body. The inflammatory and degenerative changes are chiefly found in the peripheral nerves, but lesions can also occasionally be demonstrated in the motor cells of the cord.

The extensor muscles of the forearms and fingers are weakened, causing *wrist-drop* and inability to straighten the fingers (Fig. 34).

The supinator longus escapes, the paralysis being usually limited to the distribution of the posterior interosseous branch of the musculo-spiral nerve. Though the flexors of the forearm are not paralysed, the grasp is greatly weakened owing to the inability of the extensors to fix the wrist. One hand is often affected before the other, and to a greater degree. After the dropped-wrist condition has existed for some time, a swelling appears on the back of the hand, due partly to displacement of the bones of the wrist and partly to changes in the synovial membranes. There is seldom any definite disturbance of sensation. Another variety, which may occur independently of. or in association with, the forearm distribution, is that in which the loss of power affects the small muscles of the hand. The

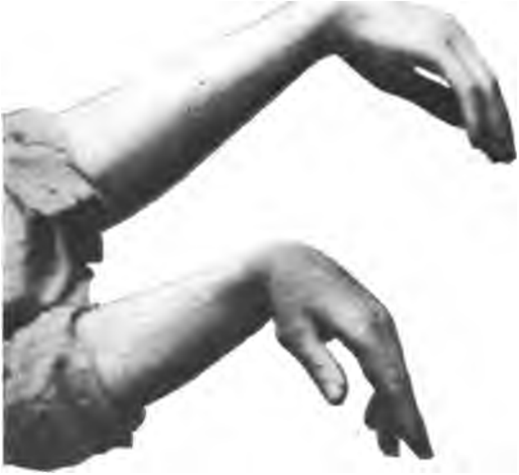

Fig. 34.—Wrist-drop in neuritis from chronic lead-poisoning.

interossei and muscles of the thenar eminence waste and the signs closely resemble those of progressive muscular atrophy. Only in severe cases does the paralysis extend beyond the forearms. In rare instances a spasm of the muscles occurs. In some cases there are symptoms of disturbance of the central nervous system, such as headache, vomiting, mental impairment, and optic neuritis; paralysis of cranial nerves has occasionally been observed. Recovery generally takes place very slowly, and the patient is liable to relapse if he again comes under the influence of the poison.

Postdiphtheritic Neuritis

Postdiphtheritic paralysis occurs in children and adults, but more often in the former. The first signs may show themselves a few days after the disease has commenced, or, on the other hand, they may not appear for several weeks; the usual period of onset is about a fortnight after infection.

Symptoms.—In the majority of cases paralysis of the soft palate is the first symptom, as evidenced by the regurgitation of fluids through the nose, and the presence of a "nasal" voice.

The ocular group of muscles is generally the next to be affected, and though any of these may be paralysed, the external rectus and the ciliary muscles are those most frequently weakened. In severe cases the paralysis is more generalized, and there may be loss of power in all the limbs.

Of particular importance is the vulnerability of the vagus and the phrenic nerves to the effects of the toxin.

The knee-jerks are generally lost early, and indeed they are often absent after an attack of diphtheria without any other symptoms of neuritis. There may be ataxy of the limbs, and sensory symptoms, such as pain, tingling, and anæsthesia, may be complained of. Vomiting is sometimes a troublesome symptom.

Observations made by Walshe* show that an initial local paresis frequently occurs at the site of the infective focus, wherever the latter may be, e.g. an infection of the thumb gave rise to a neuritis of the arm independent of the generalized multiple neuritis which followed. Walshe considers that the palatal paralysis in faucial diphtheria is a corresponding local manifestation at the site of infection, so that the complete symptom-complex in any given case would consist of (1) an initial local paresis related anatomically to the site of the infective focus, (2) paralysis of accommodation, (3) polyneuritis.

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* *Proc. Roy. Soc. Med.*, March, 1920.

Course and prognosis.—Many cases run an uneventful course, and power gradually returns. When the paralysis does not spread beyond the soft palate and the ocular muscles, recovery often takes place quickly, but when the limbs are affected the duration is much longer, and it may be several months before power is restored. Signs that the power of the heart or of the diaphragm is impaired always give the case a grave aspect. "Bulbar crises" characterized by failure of heart and respiration may occur, and these are often associated with paralysis of the diaphragm (Pasteur).

Pathology.—The toxin appears to affect the whole of the lower motor neurone, and a parenchymatous degeneration of the myelin sheath of the nerves seems to be the most important lesion found after death (F. E. Batten).

Changes in the motor nuclei of the cord and bulb have, however, also been described, and alterations in the structure of the motor cells of the anterior horns of the cord have been noted in animals at quite an early stage of diphtheria intoxication.* Walshe considers it probable that the initial local palsy is due to the passage of the toxin along the perineural lymph-channels of the peripheral nerves (*see* p. 74), the multiple neuritis being the result of a generalized blood infection.

Fatty degeneration has been observed in the muscle tissue of the heart and diaphragm, and also in other viscera, and is regarded by Dudgeon as the essential cause of the acute cardiac failure which is apt to occur in these cases.

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* A. G. R. Foulerton and Campbell Thomson, *Edin. Med. Journ.*, Jan., 1902.

A type known as **acute febrile polyneuritis** sometimes occurs. Its characteristic features, as described by Gordon Holmes,* are those of a rapidly developing widespread flaccid paralysis which often affects the muscles of the face and deglutition, with occasional involvement of the muscles of the eyes and sphincters. Sensory changes are usually slight. This variety has to be differentiated from neuritis due to other causes, and from cord lesions, e.g. acute ascending myelitis, poliomyelitis, and Landry's paralysis.

Diagnosis of multiple neuritis. — Peripheral neuritis has to be distinguished from the various forms of *myelitis.* The main distinguishing features are the signs in neuritis of a lower neurone lesion, and the absence of sphincter trouble. A transverse myelitis above the lumbar enlargement is associated with the signs of an upper neurone lesion together with loss of control over the sphincters.

Other important differences are to be found in the distribution of the anæsthesia and in the formation of bedsores.

In transverse myelitis there is anæsthesia below the level of the lesion, but in neuritis the sensory loss is confined to the limbs and does not extend to the trunk. The acute bedsores that are so commonly associated with myelitis do not occur in neuritis.

A myelitis of the lumbar enlargement may superficially resemble a peripheral neuritis, inasmuch as the knee-jerks may be lost, and the muscles of the thighs and legs may waste from destruction of the motor cells in the cord at the level of the lesion, but the complete loss of control over the sphincters which occurs in these circumstances is still a distinguishing feature.

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* *Brit. Med. Journ.*, June 14, 1917.

From *acute anterior poliomyelitis* the chief distinctions are the alterations of sensation and the symmetry of the paralysis as compared with the irregularly scattered lesions of poliomyelitis. There are, however, some cases in which it is very difficult to arrive at a differentiation.

From *Landry's paralysis* there is also a difficulty, in some cases, in distinguishing peripheral neuritis, though in characteristic examples of Landry's disease the absence of any alteration of sensation, of wasting, and of electrical changes in the muscles is a distinguishing feature.

The symptoms in certain cases of neuritis closely simulate those of *locomotor ataxy.* The resemblance occurs when incoördination is a prominent feature, and, where this exists in combination with absent knee-jerks and the presence of pains in the legs, tabes is apt to be diagnosed. Some cases of alcoholic neuritis closely resemble tabes in these particulars, as also do some cases of neuritis due to diabetes, in which the similarity may be marked by the presence of gangrenous ulcers on the feet, but the Argyll-Robertson pupil of tabes and the loss of power with the altered electrical reactions in neuritis will assist the diagnosis.

From *hysterical paralysis* neuritis can be differentiated by the absence in the latter of knee-jerks and the presence of wasting and changes in the electrical reactions of the muscles.

Prognosis.—The prognosis varies with the nature of the cause and the possibility of its removal. In general the chances of recovery of power are good if the cause can be removed, but the progress is often very slow.

In alcoholic neuritis, when the cause is removed improvement usually takes place, except in the most severe cases, in which death may occur from general exhaustion or cardiac failure.

Signs of impairment of the functions of the vagus or phrenic nerves are always grave, and the special liability of these to be damaged in postdiphtheritic paralysis has been already mentioned.

Treatment.—It is obvious that, whenever possible, the cause must be removed at once.

Little can usually be done to shorten the process of recovery, but salicylate of soda and iodide of potassium sometimes appear to have favourable effects. After the acute symptoms have subsided, strychnine is probably the most useful drug. When the pains are severe, local applications of heat and liniments containing chloroform and belladonna are useful, and occasionally alternate successive applications of heat and cold give relief. Phenacetin, antipyrin, antifebrin, aspirin, and salicylates are the drugs that are most useful as anodynes. In the worst cases recourse must be had to morphia.

Massage and electrical treatment should be commenced early, and care must be taken to prevent the paralysed limbs from assuming faulty positions, especially with regard to the feet.

In the postdiphtheritic cases it is most important to keep the patient at rest till the danger of cardiac failure is over, for fatal syncope may result from the patient merely sitting up in bed. During the attacks of respiratory failure which are apt to occur in these cases, artificial respiration may tide over the time of danger.

When lead has caused the neuritis, its elimination should be hastened by the regular administration of saline purgatives, of which magnesium sulphate is the one most often chosen. Iodide of potassium is also supposed to assist the elimination of both lead and arsenic, but the results of analyses of the excreta seem to show that it has not much influence, at any rate so far as the elimination of lead is concerned.

CHAPTER XV

TUMOURS OF NERVES (NEUROMATA)

Nerve trunks are sometimes the seat of tumours which are usually all grouped under the clinical title of neuromata, though their structure varies and is indeed seldom that of nerve tissue. Those most frequently met with are **neuro-fibromata,** which form fusiform swellings on the nerve trunks, and are very liable to undergo myxomatous degeneration. They may grow from the cranial or the spinal nerves, and are often multiple.

Plexiform neuromata (Fig. 35) are rare. They form more diffuse tumours than the neuro-fibromata and feel " like a bag containing a number of tortuous, irregular vermiform bodies, soft to the touch and mobile " (Bland-Sutton).

Closer examination in these cases shows that multiple connective-tissue tumours have formed along the course of the nerves. Of the cranial nerves the trigeminal is the most frequently attacked, but the growth may occur in any nerve, cranial or spinal.

Ganglionic neuromata are very rarely met with. They are formed of nerve cells, nerve fibres, and neuroglia.

Neuro-fibromata may arise in great numbers from the twigs of the cutaneous nerves and constitute the condition known as molluscum fibrosum, which, when associated with freckles or a more diffuse pigmentation of the skin, is known by the name of von Recklinghausen's disease.

Symptoms.—The symptoms depend largely on where the tumours happen to be situated. Growths on sensory nerves may cause pain and various disturbances of sensation; among the most painful are those which arise on the nerve-endings of an amputation stump.

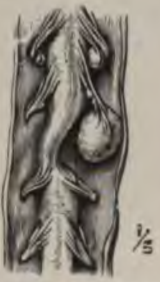

Fig. 36.—Cervical segment of the cord represented in Fig. 35. A nodule on one of the cervical roots compressed the cord and led to fatal paraplegia. (*Sir J. Bland-Sutton.*)

Pressure on spinal roots may cause weakness in the corresponding muscles, which may be followed later by signs of paraplegia as the tumour invades the spinal cord (Fig. 36).

Prognosis.—Tumours growing from nerves are

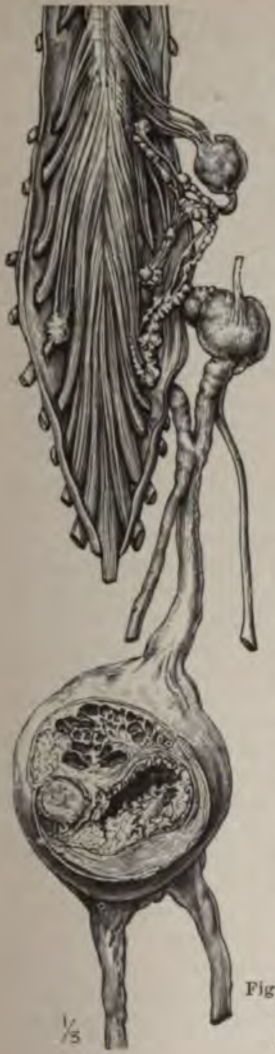

Fig. 35.—Plexiform neuromata affecting the roots of the cauda equina and anterior crural nerve. (*Sir J. Bland-Sutton.*)

Treatment.—When the tumo
causing unpleasant symptoms it sho
when the growths are multiple the dif
greater, and in the molluscum fibro
can be done.

SECTION III.—MUSCULAR DYSTROPHIES

CHAPTER XVI

THE MYOPATHIES; AMYOTONIA CONGENITA; PERONEAL TYPE OF ATROPHY

THE MYOPATHIES

UNDER the term "myopathies" is comprised an important group of cases in which the failure of vitality appears to arise in the muscles themselves. The essential feature of these cases is atrophy of the muscles, with which, however, there may be some accompanying enlargement. In many of the forms there are no definite changes to be found in the nervous system, but in some varieties, e.g. the peroneal type, a neuronic degeneration is associated with the atrophy.

Morbid anatomy.—Atrophy of some fibres, pseudo-hypertrophy of others, and excess of fat and fibrous tissue are the principal changes observed in the affected muscles. While all these cases seem to be pathologically similar in their want of vitality, they vary considerably in their clinical appearances. Among the forms most commonly met with are:

1. The pseudo-hypertrophic form.
2. The facio-scapulo-humeral form, known as the Landouzy-Dejerine type.
3. The juvenile form of Erb.

There have been described also a "distal" form, in which the atrophy begins in the extremities of the limbs, and a "myotonic" form, in which atrophy is associated with tonic contractions of the muscles that relax slowly after voluntary movements. Amyotonia congenita and the peroneal type of atrophy are also thought to be closely allied to this group.

As might be expected from their common pathological basis, no sharp line can be drawn between the different forms. Cases in which atrophy is the dominant feature often show traces of pseudo-hypertrophy, while cases of pseudo-hypertrophy almost invariably show some signs of atrophy.

Like the parallel cases of spinal origin, the myopathies are very liable to occur in several members of a family.

1. PSEUDO-HYPERTROPHIC PARALYSIS

Etiology.—This disease generally begins between the ages of 4 and 10. It exhibits a distinct tendency to run in families, and is usually transmitted by females and inherited by males.

Symptoms.—Clumsiness, frequent falls, and a difficulty in going upstairs are generally among the early symptoms, all of which seem the more strange to the parents since the limbs, especially the calves, often appear to them to be unusually well developed. Examination, however, shows that the legs are weak in spite of their increased size, the latter proving to be a pseudo-hypertrophy due to overgrowth of fat and connective tissue which have displaced the wasted muscle fibres.

Distribution of hypertrophy.—The muscles in which some enlargement can most usually be seen are those of the calf (Fig. 37), the quadriceps extensor, the glutei, the triceps, and the infraspinati. They are hard and firm to the touch, but despite this they are weaker than they should be. In some cases part of a muscle is enlarged and part atrophied.

Muscles most commonly atrophied.—Some muscles are from the first atrophied and show no sign of enlargement; among them the most constant are the lower half of the pectoralis major and the latissimus dorsi. Others in which atrophy can very frequently be detected are the biceps, the flexors of the knee, and the peronei (Fig. 37).

Certain muscles generally *escape*, e.g. the muscles of the face, forearms, and hands, but occasionally some enlargement of the tongue may be observed.

Attitude.—The failure of muscular power makes it difficult for the patient to stand, walk, or rise from the ground, and, in endeavouring to perform these actions, fairly characteristic attitudes are assumed. In standing the feet are kept wide apart in order to obtain a broader base, while the shoulders are thrown back (lordosis) in order to keep the line of gravity from falling too far forwards. The arms are generally flexed and adducted.

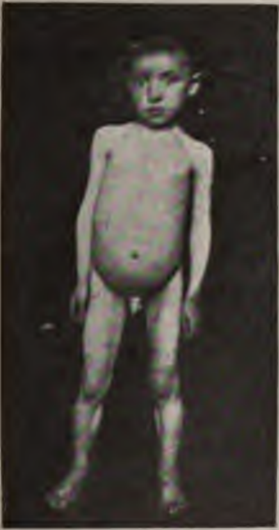

Fig. 37.—Case of pseudo-hypertrophic paralysis. (*Pierre Marie.*) Observe the enlargement of the muscles of the thighs and calves as compared with the thin arms.

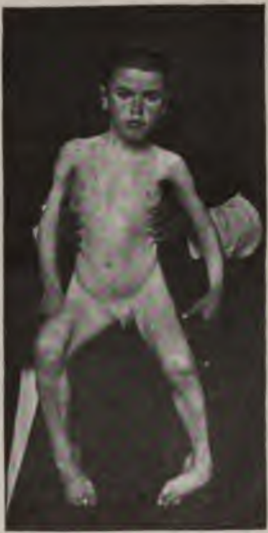

Fig. 38.—Deformities arising in the course of pseudo-hypertrophic paralysis. (*Pierre Marie.*) This is the brother of the patient in Fig. 37.

In walking the same position is preserved, and the patient waddles from side to side, with the feet turned out, and with a "high-stepping" gait. (Plate 7.)

In rising from the supine position the patient first rolls over, then takes his fixed points from his hands and toes, and finally helps himself up by placing his hands on his thighs.

All these positions are due to attempts to compensate for the weakness of the affected muscles, and therefore they vary widely in degree in different cases.

Reflexes.—Wasting of muscles leads to the loss of any reflexes which they may normally serve; thus the knee-jerk diminishes as changes occur in the quadriceps extensor muscle.

Deformities.—Talipes of the feet and curvature of the spine are both often met with. (Figs. 38, 42.)

2. FACIO-SCAPULO-HUMERAL TYPE

In the facio-scapulo-humeral type (known also as the Landouzy-Dejerine form) the atrophy, like that of pseudo-hypertrophic paralysis, generally begins in childhood. It is often spoken of as the "infantile form" of myopathy in contradistinction to the "juvenile form," which begins in later life.

The name "facio-scapulo-humeral" shows the chief distribution of the wasting. (Fig. 39.)

In the face the orbicularis palpebrarum and the orbicularis oris are commonly weakened, and as a result of the weakness of the latter muscle, together with others at the corner of the mouth, quite a characteristic expression is given to the features.

Next, the muscles of the shoulder-girdle and upper arm atrophy, and here the trapezius (both upper and lower divisions), latissimus dorsi, serratus magnus, pectorals, triceps, and biceps generally suffer early. The wasting of the serratus magnus causes the vertebral border of the scapula to tilt whenever the arm is lifted.

The forearms generally escape, the hands always.

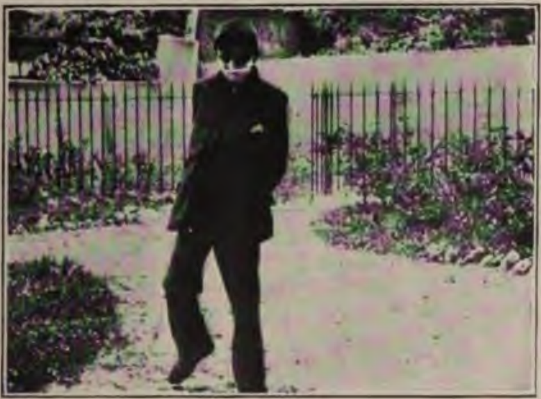

PLATE 7. — Instantaneous photograph showing "high-stepping" gait in pseudo-hypertrophic paralysis.
(By courtesy of Dr. George Ogilvie.)

From the shoulder-girdle the wasting spreads to other muscles of the trunk and limbs. The muscles of the back and hip become weak, causing lordosis and a waddling gait like that in the pseudo-hypertrophic form, while wasting of the other muscles is accompanied by difficulty in walking and deformities of the feet.

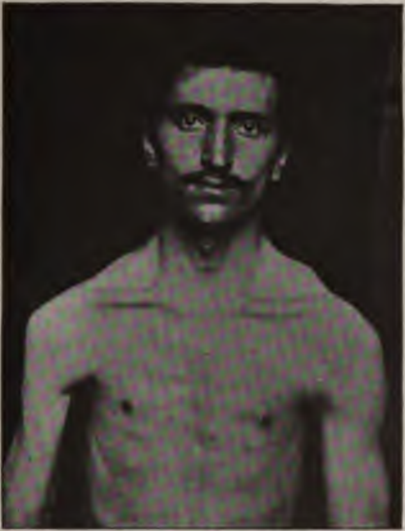

Fig. 39.—Appearance of neck produced by displacement of the scapulæ and dropping of the shoulders in a case of myopathy. (*Pierre Marie.*)

Some muscles or parts of muscles are often found to be enlarged (Fig. 40), and thus a relationship is established between this and the pseudo-hypertrophic form.

3. Erb's Juvenile Form

In the juvenile form the wasting is generally noticed rather later in life, but nearly always before the patient is out of his teens. The wasting generally begins about the shoulders and then spreads to the trunk (Fig. 41) and legs; the muscles of the face are also sometimes affected. Enlargement of some muscles may be observed.

Diagnosis of the myopathies.—From progressive muscular atrophy of spinal origin the chief points of distinction are:

i. The age at which the disease begins.

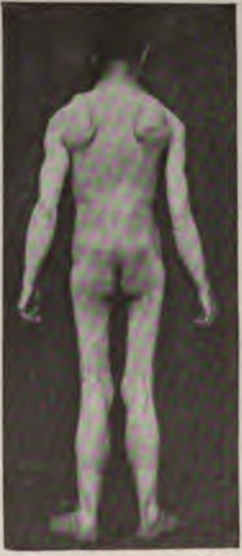

Fig. 40.—Case of myopathy showing enlargement of the infraspinati. (*Pierre Marie.*)

ii. The distribution of the wasting. In the myopathies now under consideration the muscles of the trunk waste early, the hands usually escape, and the deltoid is often normal; whereas in progressive muscular atrophy of spinal origin the hands and also the

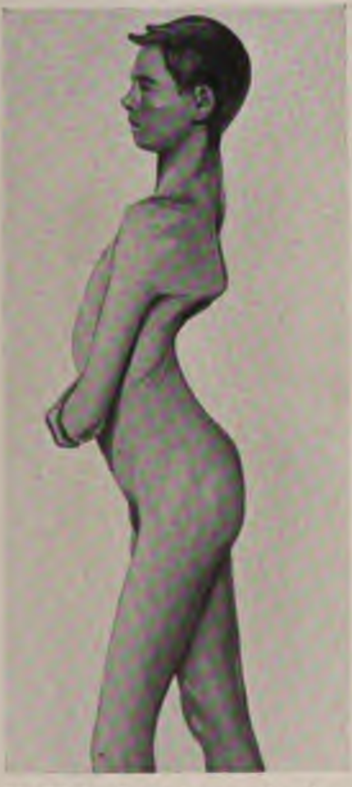

PLATE 8.—Case of myopathy showing the characteristic attitude of lordosis.

deltoid are among the earliest parts to exhibit signs of the disease.

iii. The myopathies tend to occur in several members of a family, and when this peculiarity is present the spinal variety may be excluded.

iv. Hypertrophy, either true or false, is not found in the cases of spinal origin.

v. Fibrillary tremors are common in the spinal cases and rare in the myopathies;

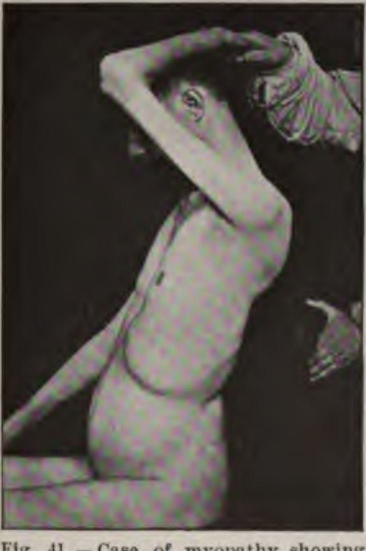

Fig. 41. — Case of myopathy showing wasting of the muscles of the trunk causing the deformity of the trunk and the waist known as the "*taille de guêpe.*" (*Pierre Marie.*)

The distribution of the wasting, absence of sensory changes, and character of the electrical reactions serve to differentiate these cases from peripheral neuritis.

The electrical changes shown in the myopathies are usually those of diminished reactions to both the faradic and galvanic currents. There is generally no definite "reaction of degeneration."

Prognosis.—All these different forms of the myopathies tend to progress slowly, but here and there arrest takes place or the rate of progression is so slow that it can be ignored.

The danger to life lies in the atrophy extending to the muscles of respiration, and consequently in the failure to combat successfully any pulmonary complications such as bronchitis, pneumonia, and tubercle.

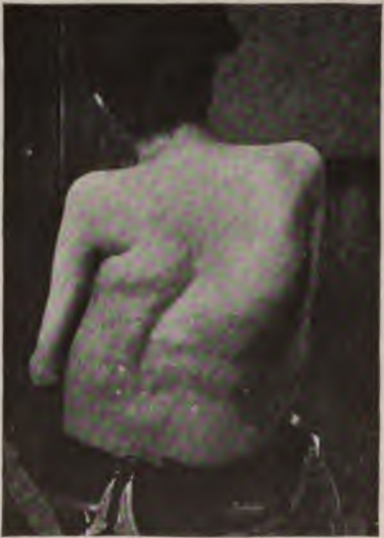

Fig. 42.—Curvature of the spine occurring in the course of myopathy. (*Pierre Marie.*)

Treatment.—Every effort to arrest the wasting must be made. Massage, moderate exercise, and electricity should all be used to keep up the nutrition of the muscles, and must be persevered with if any success is to be obtained.

AMYOTONIA CONGENITA (MYATONIA CONGENITA)

In contradistinction to the condition known as myotonia congenita, described at p. 472, there is an uncommon condition of obscure origin, which was first described by Oppenheim, and to which the title of amyotonia (or myatonia) congenita has been given. The condition appears to be associated with myopathy in some cases, while in others the connection is not so clear. It also occurs in the atonic form of cerebral diplegia of children.

Etiology.—The disease is met with in children, and in the majority of cases has either existed from the time of birth or come on very shortly afterwards. In a few the history gives rise to suspicions that the trouble has followed some illness, e.g. measles in early childhood, but it is possible that the condition has pre-existed, and only become more obvious through the illness.

Symptoms.—The characteristic features in these cases are weakness and absence of muscle tone, so that the limbs can be placed in all kinds of grotesque attitudes. (Figs. 43, 44.)

The distribution of the disability varies in different cases. In some cases the face, trunk, and limbs may all be affected, but in others the muscles of the limbs, and especially of the legs, are the only parts which lose their tone.

The power of movement is greatly diminished, so that the child cannot stand or walk, and when the trunk muscles are toneless it may be unable even to sit up. If the face is affected there may be inability to close the eyes.

The muscles in the diseased areas are flabby, and a faradic current of unusual strength is necessary to make them contract; moreover, currents of far greater intensity than can be borne by a normal child appear to cause no pain.

Diagnosis.—The principal diseases from which amyotonia has to be differentiated are rickets (with which it may, of course, be associated), anterior poliomyelitis, and possibly the myopathies.

From *anterior poliomyelitis* it is distinguished by its symmetrical distribution and by the absence of complete paralysis, for in amyotonia the movements, while they are weakened, are not lost. Further, there is not the wasting which occurs in acute anterior poliomyelitis.

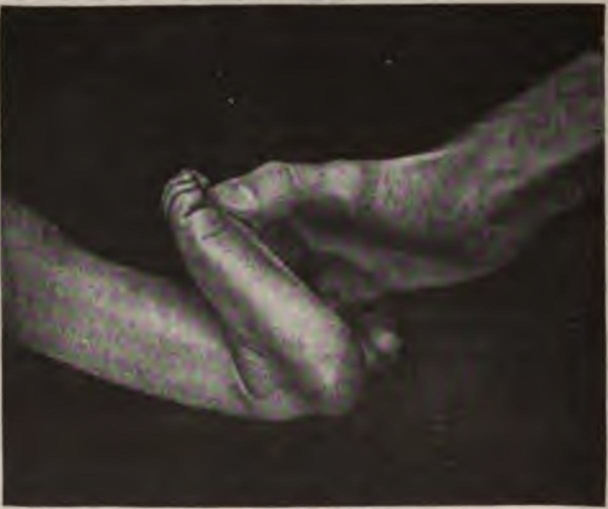

Fig. 43.—Extreme flexion of the ankle-joint in amyotonia congenita. (*From a case under the care of Dr. Essex Wynter.*)

With the *myopathies* this condition has much in common, and, as already mentioned, some cases are regarded as a variety of that group, while in others lack of familial tendency, gradual improvement, and the absence of pronounced wasting are relied upon as being characteristic features of distinction between the two diseases.

Prognosis.—The prognosis, as judged from the cases hitherto recorded, appears to be favourable. The muscles improve as the child grows, and eventually they become stronger. In some instances contractures have been observed.

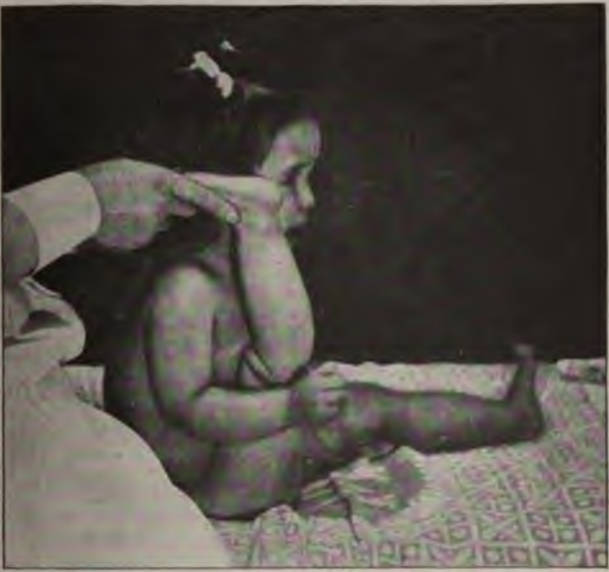

Fig. 44.—Grotesque position of the leg in the same case as Fig. 43.

Treatment.—The main points in treating these cases are: (1) to look after the general nutrition; (2) to regulate the child's movements so that deformities and ill-shapen limbs are not allowed to develop from attempting to walk too soon; (3) to massage and move the limbs in order to prevent the formation of contractures and fixed positions.

THE PERONEAL TYPE OF MUSCULAR ATROPHY

The early descriptions of this variety, which is characterized by wasting of the muscles of the legs and forearms, are especially associated with the names of Charcot, Marie, and Tooth.

Etiology.—Nothing definite is known of the causation. The symptoms generally first appear in children or young adults. There is a tendency for several members of a family to be attacked, and cases of heredity have occasionally been recorded.

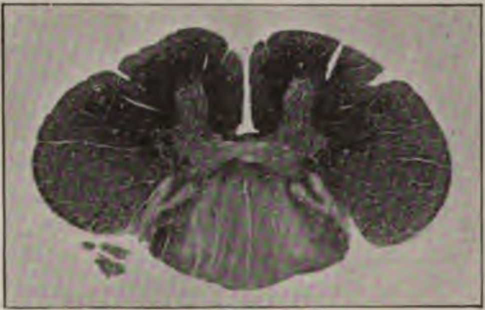

Fig. 45.—Section of spinal cord showing degeneration of the posterior columns, from a case of peroneal atrophy. (*Pierre Marie.*)

Pathology.—The exact pathology is still doubtful, and in the light of present knowledge it is convenient to look upon this variety as an intermediary form between the atrophies of muscular and those of spinal origin. Sclerosis of the posterior (Fig. 45) and, to some extent, of the lateral columns of the cord has been observed, and the occurrence of a curious hypertrophic form of neuritis has been occasionally noticed.

Symptoms.—The onset is very gradual. At first the peronei and extensors of the toes become weak, and later on the calf muscles waste, so that the size of the

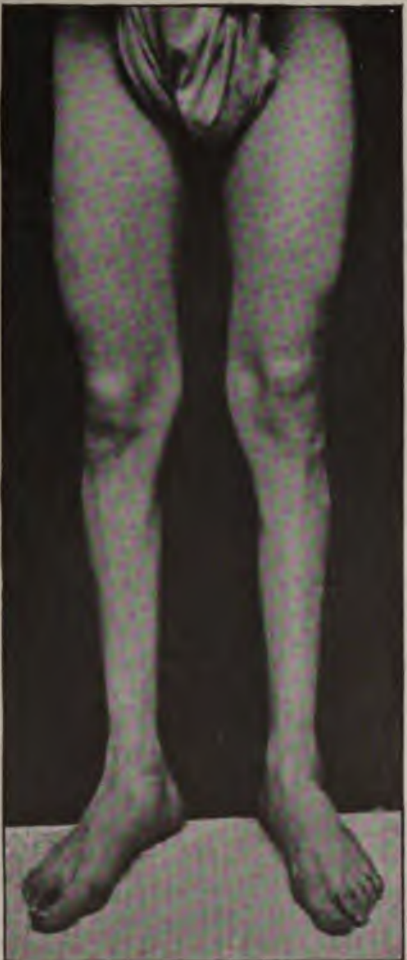

Fig. 46.—Case of peroneal atrophy. Note the wasting of the muscles below the knee.

leg contrasts markedly with that of the thigh, which generally remains normal for some time (Fig. 46). Clubbing of the feet occurs, and is often an early sign.

When the thigh muscles remain normal the knee-jerks may be retained. After a time, wasting takes place in the small muscles of the hand and spreads to the muscles of the forearm. Deformities resulting in the "claw hand" may eventually arise. In some cases the disease does not make any further progress, but in others the wasting gradually spreads to the thigh and trunk.

Disturbances of sensation are uncommon, but pain and anæsthesia of the limbs have occasionally been observed.

Diagnosis.—The main points upon which reliance has to be placed are slow symmetrical wasting of the legs, accompanied by clubbing of the feet, followed later by wasting and deformities in the hands and forearms, occurring in children and young adults, though from what has already been said it will be seen that cases may occur in which there is doubt about the underlying spinal and neural changes.

Prognosis.—The prognosis as regards recovery is bad, but as regards life it is more favourable as compared with the other forms of myopathy, for in the peroneal type the atrophy often ceases to extend.

Treatment.—Massage, electricity, tonics, and attention to the general health are the only methods of treatment applicable to this disease.

SECTION IV.—DISEASES OF THE SPINAL CORD

CHAPTER XVII

CLASSIFICATION

THE spinal cord begins at the lower end of the medulla and terminates at about the upper border of the 2nd lumbar vertebra, below which are the nerve roots of the cauda equina.

In practice the patient comes with certain complaints which have to be investigated and referred to their proper causes. But before this can be done it is necessary that some knowledge should be acquired of the principal lesions which are likely to be met with, and for the purpose of gaining this knowledge classifications are utilized.

It is true that such classifications are necessarily often artificial and not altogether scientific, but they nevertheless form a useful working basis and serve as an aid to memory, while they can do but little harm so long as their provisional nature is duly emphasized.

One such method of dealing with diseases of the spinal cord is to classify them according to the anatomical distribution of their lesions, and, taken in this way, the disorders at once separate themselves into two large groups, viz.:

1. Those in which the morbid changes are confined within the limits of certain columns, i.e. the so-called **system lesions**; and
2. Those in which the morbid changes are distributed irregularly through the substance of the cord quite irrespectively of column formation, i.e. **non-system lesions.**

System Lesions

System lesions are produced by neuronic degeneration. The separate columns of the cord (Plate 9) are composed of bundles of neurones, and stress has already been laid on the fact that every individual neurone of the bundle is a unit in itself, and dependent on its own cell body for its nutrition.

When this nutrition is impaired, the neurone dies and its branches wither away just as do the branches of a tree when something interferes with its roots.

Certain poisons, of which that of syphilis is one of the most important, cause neuronic degeneration, and the symptoms in any given case depend upon the particular system or systems of neurones affected.

Reference to the accompanying diagrams shows the chief systems, and these may be attacked singly or in numbers, and accordingly there may be single or combined system lesions.

Of the **single-system lesions,** where only one column is attacked, we may recognize the following:

1. Disease of the cells of the *anterior horns,* as exemplified in acute poliomyelitis and progressive muscular atrophy (Fig. 47).
2. Disease of the *lateral* (crossed pyramidal) *tracts,* as exemplified in lateral sclerosis (Fig. 48).
3. Disease of the *posterior columns,* as exemplified in locomotor ataxy (Fig. 49).

Multiple-system lesions may occur as follows:

1. Disease of the *lateral columns* together with that of the *cells of the anterior horns,* as exemplified in amyotrophic lateral sclerosis (Fig. 50).
2. Disease of the *lateral columns* together with that of the *posterior columns,* as exemplified in ataxic paraplegia (Fig. 51).
3. Disease of the *lateral columns* together with that of the *posterior columns, cerebellar tracts,* and (sometimes) *antero-lateral tracts,* as exemplified in Friedreich's disease and subacute combined degeneration (Fig. 52).

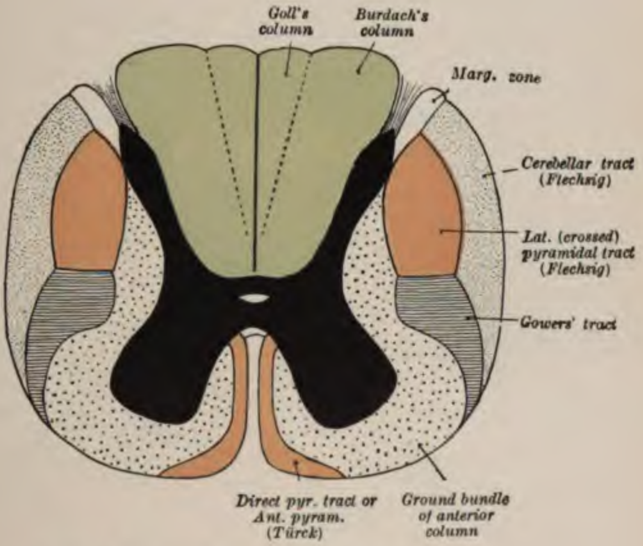
Goll's column
Burdach's column
Marg. zone
Cerebellar tract (Flechsig)
Lat. (crossed) pyramidal tract (Flechsig)
Gowers' tract
Direct pyr. tract or Ant. pyram. (Türck)
Ground bundle of anterior column

PLATE 9.—Tracts of the spinal cord. (*Morat.*)

Thus the groundwork of the combined lesions making up the clinical signs of amyotrophic lateral sclerosis and ataxic paraplegia can be deduced, without any special

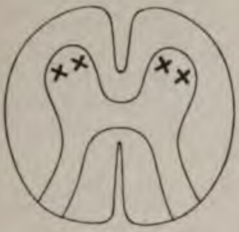

Fig. 47.

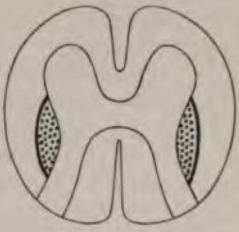

Fig. 48.

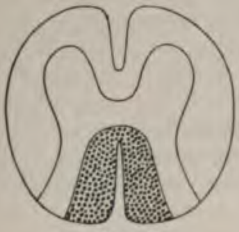

Fig. 49.

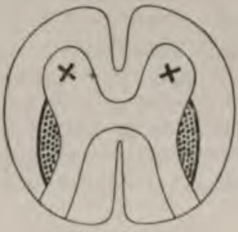

Fig. 50.

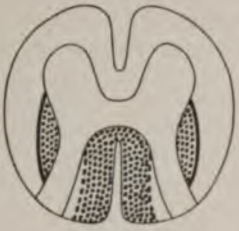

Fig. 51.

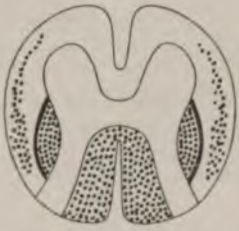

Fig. 52.

effort of memory, by combining the symptoms of lateral sclerosis of the lower limbs with those of progressive muscular atrophy of the upper limbs in the case of the first, and with those of ataxia in the case of the second.

Irregular or Non-System Lesions

In these no special predilection is shown for the various columns, and the classification has to be based on the pathological possibilities. The main lesions are:

Inflammations, e.g. myelitis—transverse or disseminated.
Vascular lesions, e.g. thrombosis and hæmorrhage.
Diffuse and disseminated degenerative processes, e.g. syringomyelia and disseminated sclerosis.
Syphilitic lesions.
Tumours.
Pressure from without, as in spinal caries, meningeal disease, injuries, etc.

This method of classification is, of course, very far from perfect. For instance, the degeneration in tabes dorsalis is not entirely confined to the posterior columns. Nevertheless the classification is one that proves useful to students, since it serves as a model which they can supplement or vary at their discretion, and at the same time it helps to a considerable extent to demonstrate the relationship which exists between the different diseases.

CHAPTER XVIII

PARAPLEGIA AND BULBAR PARALYSES

PARAPLEGIA

PARAPLEGIA is the clinical term usually applied to a condition of paralysis of both legs, though it is equally applicable when the arms are also involved, as, for instance, in a case of fracture of the spine in the cervical region.

The presence of paraplegia indicates that the transmission of impulses along the motor fibres has been interrupted, and its causes are best studied by making a diagram of the motor paths and then considering the whereabouts and nature of the possible interruptions.

1. **Lesions of the peripheral nerves.**—Peripheral neuritis from alcohol, arsenic, or any other cause may give rise to complete loss of power in both legs, and the position of the lesion has to be decided by the characteristics of lower neurone paralysis, and then differentiated from diseases of the anterior cornua.

2. **Lesions of the spinal cord.**—Disease of the cells of the **anterior horns** in the lumbar region will, if symmetrical, cause paraplegia.

Here the paralytic symptoms, being of the lower motor neurone type, are similar to those of peripheral neuritis, but there will be no sensory changes such as occur when the peripheral nerves are affected. As a matter of fact, a paraplegia of symmetrical distribution due to a lesion limited to the cells of the anterior horns is rare. In poliomyelitis a symmetrical lesion is unusual, while in progressive muscular atrophy degeneration of the motor cells of the lumbar region is seldom a predominant feature.

Any lesion of the spinal cord which implicates the **lateral columns** (crossed pyramidal tracts) will cause some degree of paraplegia. These may be divided into:

(*a*) Those which occur primarily in the substance of the cord.

(*b*) Those which press on the cord from without.

(*a*) Those of the *substance of the cord* may be transverse lesions (complete or incomplete), such as arise from myelitis, hæmorrhages, thrombosis, tumours, and syringomyelia, or they may be system lesions in which definite columns are attacked, as in primary lateral sclerosis, ataxic paraplegia, and subacute combined degeneration.

(*b*) *Pressure from without* may be due to fracture-dislocation of the spine, caries, or tumours.

In all these cord lesions, except when the lumbar enlargement is diseased, the paralysis of the legs is that of the upper neurone type, as shown by the increased tendon reflexes, rigidity, ankle-clonus, and extensor response of the plantar reflex, while there are no wasting (except from disuse) and no changes in the electrical reactions of the muscles. The action of the bladder varies; when the lesion extends across the cord there is usually trouble, but in some of the system lesions little or nothing may be noticed.

3. **Lesions of the brain.** (*a*) **Subcortical.**—As we pass above the cord the motor fibres diverge to the cortex of the brain, and therefore between the region of the pons and the cortex it is unlikely that any single lesion would injure them on both sides. (Plate 1.)

Symmetrical lesions are naturally uncommon, hence hemiplegia rather than paraplegia is the usual result of disease in this area; but it does occasionally happen that two vascular lesions succeed one another and cause a "double hemiplegia," or "diplegia," which is to all intents and purposes a paraplegia.

(*b*) **Cortical.**—In the cortex the motor areas for the legs are not far from each other, and any injury to the vertex of the brain is liable to damage both the leg centres and cause paraplegia. Some of the paraplegias (known as diplegias) of infancy arise in this way through injuries at birth (*see* Cerebral Diplegia, p. 330), but it is seldom that paraplegia arises in this manner in later life.

(*c*) **Hysteria.**—Paraplegia of a most complete type may be of hysterical origin, and its cause is then probably situated in the cerebral cortex.

Some of these hysterical cases give rise to great difficulty in diagnosis, for though they can usually be easily distinguished from lower neurone lesions, such as neuritis, where the wasting and the loss of reflexes make the condition clear, they are often confused with cord lesions, and especially with those due to commencing disseminated sclerosis, in which the symptoms are apt to vary in the same extraordinary fashion as they do in functional disease.

It is in these doubtful cases that the extensor response of the plantar reflex is so valuable, for increased knee-jerks, with perhaps slight ankle-clonus, may be present in both.

Further particulars of differential diagnosis of disseminated sclerosis will be found at p. 216.

For the **prognosis** of paraplegia reference may be made to the chapters dealing with the different diseases in which paraplegia occurs.

SENILE PARAPLEGIA

In elderly people some weakness of the legs, accompanied, perhaps, by difficulty in exercising full control over the sphincters, may occur from anæmia of the cord due to atheroma of the spinal arteries.

INTERMITTENT CLAUDICATION

Another difficulty in walking—also due, it is supposed, to atheroma—is that known under the name of *intermittent claudication*, or *limping*, and characterized by sudden attacks of lameness, often accompanied by cramps, blueness of the extremities, and sensations of tingling. After walking a variable distance the patients are pulled up suddenly with cramp, which usually passes off with rest. The symptoms in these cases are frequently attributable to impaired nutrition of the muscles consequent on the arterial disease, and absence of arterial pulsation and the presence of gangrene in the feet may be associated conditions. In other instances the trouble may be secondary to lesions of the cord or brain.

BULBAR PARALYSES

Paralyses of bulbar origin, in which the tongue, pharynx, and larynx are affected, may, like other paralyses, be of upper or lower neurone type. (Fig. 53.)

1. **Upper neurone type.**—In this form the motor fibres are interrupted in their course from the cortex to the medulla, but, in order to produce any permanent effect on the bulbar functions, the interruption must be bilateral. A unilateral lesion, e.g. a hæmorrhage into the internal capsule, though it may affect the movements of one half of the tongue and palate, will not as a rule cause any permanent loss of articulation and difficulty in swallowing. On the other hand, a bilateral lesion, by cutting off the motor fibres from both sides of the cortex, will deprive the nuclei of voluntary innervation, and in such a case the movements of the tongue, palate, and larynx may be greatly impaired.

Bilateral lesions of cortical origin may be seen in severe cases of cerebral diplegia in children; they also occur in amyotrophic lateral sclerosis and in cases where a hæmorrhage in the region of the internal capsule is followed by another in a similar position on the opposite side. The bulbar symptoms due to upper neurone lesions are often termed *pseudo-bulbar paralysis,* to distinguish them from the lower neurone lesions in which the functions of the nuclei themselves are lost.

The same rules for upper motor neurone lesions apply here as elsewhere. The power of voluntary movement is diminished. There is inability to pout the lips in order to blow or whistle, the tongue cannot be protruded or thrust into the cheeks, and articulation is very indistinct. Mastication and swallowing become difficult, the saliva dribbles out between the paralysed lips, and, in swallowing, fluid may regurgitate through the nose. The reflexes of the palate, pharynx, and larynx are preserved, a jaw-clonus may be present, and there are no wasting and no changes in the electrical reactions of the paralysed muscles.

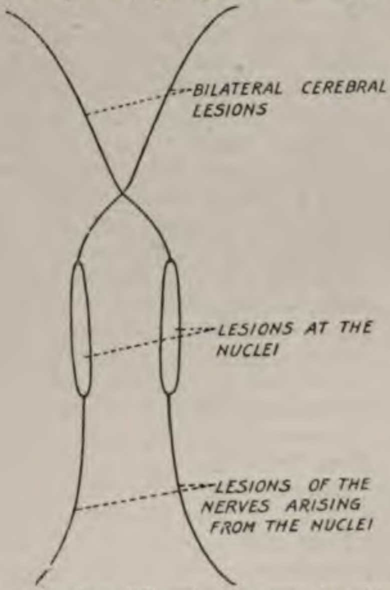
BILATERAL CEREBRAL LESIONS
LESIONS AT THE NUCLEI
LESIONS OF THE NERVES ARISING FROM THE NUCLEI

Fig. 53.—Diagram showing different situations in which bulbar paralyses may be produced.

2. **Lower neurone lesions.**—Here the disease may be in the nuclei or in the nerves arising from them. The nuclei of the bulb, from which the 10th, 11th, and 12th nerves arise, are subject to the same diseases as nuclei elsewhere. They may be grouped as follows:

(1) **Inflammations.**—Acute inflammatory affections of the bulbar nuclei are uncommon, but acute polioencephalitis, which is a condition analogous to that of anterior poliomyelitis (infantile paralysis) in the cord, and lethargic encephalitis may both occur in this region.

(2) **Degenerations.** — Chronic degeneration of the bulbar nuclei constitutes the disease known as progressive bulbar paralysis. This condition is in reality a part of progressive muscular atrophy, and is described under the heading of that disease (Chapter XXIV., p. 219).

(3) **Vascular lesions.**—(*a*) Hæmorrhage, (*b*) thrombosis, (*c*) embolism. Hæmorrhage of any size into the bulb naturally soon causes death, but thrombosis or embolism, more especially thrombosis of some small branches, may cause symptoms of disease of the bulbar nuclei. The more sudden onset of the symptoms serves to distinguish such cases from the progressive bulbar variety; their course depends upon the temporary or permanent effects which the lesion produces.

(4) **Pressure.**—Tumours in the neighbourhood of the medulla may give rise to bulbar symptoms from pressure.

When the bulbar paralysis is due to injury of the nerves after they have left the nuclei the symptoms are generally unilateral; moreover, the spinal portion of the spinal accessory will in all probability be also injured, in consequence of which the additional symptoms of paralysis of the sterno-mastoid and trapezius will be present.

Examples of neuritis in which the symptoms are symmetrical and of bulbar distribution are met with in cases of postdiphtheritic paralysis. Another disease in which bulbar signs are also often prominent is myasthenia gravis, the pathology of which is at present not certainly known, though there is some evidence to show that the morbid changes may possibly commence in the muscles themselves (*see* Chapter XLIII., p. 421).

CHAPTER XIX

ACUTE POLIOMYELITIS (INFANTILE PARALYSIS)

ACUTE poliomyelitis (known clinically also as infantile paralysis) is the result of acute inflammatory changes taking place in the motor cells of the anterior horns of the spinal cord.

Etiology.—Acute poliomyelitis is a contagious disease which may spread from person to person and may occur sporadically or in epidemic form.

The precise method by which the contagion is conveyed is still uncertain, but there is evidence to show that it can be carried by healthy persons, so that the infected patient need not necessarily have been in direct contact with anyone suffering from the disease. Most of the epidemics occur in the summer and autumn, showing that the virulence of the infection is subject to seasonal influence. Flexner and Lewis,* following the earlier experiments of Landsteiner and Popper, succeeded in transmitting the disease to monkeys, and as the result of these and other experiments it has been shown (1) that the virus has a special predilection for the spinal cord; (2) that its infective power shows a considerable resistance to glycerin and to degrees of temperature that usually affect poisons of a similar nature; and (3) that it is one of those organisms which pass through bacterial filters.

Hitherto all attempts to obtain a culture of the specific organism have failed, though more recently Noguchi is said to have isolated it.

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* "Epidemic Poliomyelitis." New York, 1910.

The length of the incubation period is uncertain, but it is thought to vary from three to ten days. The disease is especially liable to attack the young, and although adults are by no means immune the statistics of epidemics show that it is mainly a disease of childhood.

As already mentioned, the virus tends to affect the spinal cord, and it specializes this tendency by the manner in which it attacks the motor cells of the anterior horns. Careful study of the symptomatology as it occurs in large epidemics has, however, shown that the effects of the poison are by no means always confined to this part of the cord, and so it has been necessary to broaden the clinical conception of this disease and to recognize that the virus which is common to many cases does not produce in all of them the symptoms that were formerly thought to be characteristic.

Since in these epidemics the symptoms of different cases are apt to differ in such a manner as to make it difficult to form them into a common group of what was clinically recognized as acute poliomyelitis, it has been proposed to term the epidemic form Heine-Medin's disease, after Heine who gave one of the earliest descriptions of the disease, and Medin who made important observations on its epidemic form.

Pathology.—In an acute case, redness and swelling of the grey matter of the spinal cord can often be seen with the naked eye. Microscopically, acute hyperæmia, inflammatory exudation, and disintegration of the motor cells of the anterior horns are among the principal changes observed. The cell bodies are swollen, their Nissl granules broken up, and their nuclei displaced or disorganized.

Capillary hæmorrhages, thrombosis of vessels, and œdema of the cord may also be observed.

The changes are generally most intense in the grey matter of the anterior horns, but they may often be seen to extend into the posterior horns and occasionally even into the white matter.

There is also infiltration of the pia mater, and this probably explains the presence of the meningeal symptoms that are found in many cases.

The route by which the virus enters the body is not known, but once in the system there is, from experimental evidence, good reason to believe that it reaches the cord through the lymphatics and not through the blood, and while the special vascular supply of the anterior horns may have some influence in determining the localization of the disease, it is evidently not the deciding factor, as it was formerly thought to be. It is generally conceded that thrombosis and embolism are secondary effects. The cerebro-spinal fluid is sometimes clear and at other times shows lymphocytosis.

Enlargement of the spleen and swelling of the lymphoid tissue of the alimentary canal may occur, and are probably the results of a generalized infection, though by some the alimentary changes are considered as a possible indication of the path of entry of the virus.

In the later stages, after the vascular disturbance has subsided, the chief features are disappearance and disintegration of motor cells, with an excess of connective tissue that has taken their place.

Symptoms.—The early symptoms are those of a general toxæmia. The child becomes fretful and restless, and in some cases drowsiness is a prominent feature. The temperature is raised and convulsions have been known to occur, but they are unusual unless encephalitis is also present. The child dislikes being touched or moved, and seems to be in pain. Sweating and diarrhœa are often present. In the course of a day or two it is noticed that one or more of the limbs are not being freely used. This generally gives rise to alarm and leads to the examination of the child, when the most characteristic feature of this stage of the disease—presence of a flaccid paralysis of acute onset and irregular distribution—is revealed.

Groups of muscles are picked out irregularly in different limbs, but the lower limbs are affected more frequently than the upper, and the quadriceps extensor, tibialis anticus, and peronei seem especially prone to be attacked. The reflexes in the affected areas are generally lost. The muscles of the trunk, including the intercostals, may be paralysed (Fig. 54), and the respiratory movements may be still more seriously impaired through paresis of the diaphragm. It is not uncommon to find that muscles supplied by the cranial nerves are affected; and a bulbar type in which the nuclei of the medulla are affected may also occur.

It soon becomes apparent that the paralysed muscles are rapidly wasting, and at the end of ten days or a fortnight their electrical reactions, if tested, will probably be those of degeneration.

In a typical case there is no disturbance of sensation and usually no loss of control over the sphincters.

In those cases where the effects of the virus are not confined to the cells of the anterior horn the symptoms do not conform accurately to the average type. This is the explanation of cases in which some alteration in sensation has been observed, and of others in which the pyramidal columns have been affected in addition to the anterior-horn cells, but such instances are uncommon and must be regarded as exceptional.

Clinical varieties.—Many clinical varieties are met with, according to the parts of the cord that are attacked.

One of the most important is the form which resembles Landry's paralysis, with which indeed, in some cases at least, it may be identical. Here the paralysis spreads from the feet upwards with no sensory disturbance and with no appreciable wasting of muscles, giving all the appearance of greatly impaired function of the motor cells stopping short of their actual destruction.

There is also a form which closely resembles polyneuritis, and in other instances there may be symptoms of ataxy.

The virus may also attack the brain and produce encephalitis, a description of which is given at p. 341.

Diagnosis.—The acute onset with rise of temperature and wasting of muscles is very characteristic of the disease. When there is much pain the symptoms may at first resemble those of *spinal meningitis*, but as the disease progresses and wasting ensues the difference usually soon becomes apparent. As the febrile symptoms pass off, loss of power becomes the prominent feature and the wasting of the muscles and grouping of the paralysis, together with changes in the electrical reactions, show that it is the lower motor neurone which is at fault, i.e. that the lesion is situated somewhere between the cells of the anterior horns and the muscles. As acute diseases of the muscles, giving rise to such symptoms, are unknown, the issue is in most cases further narrowed between diseases

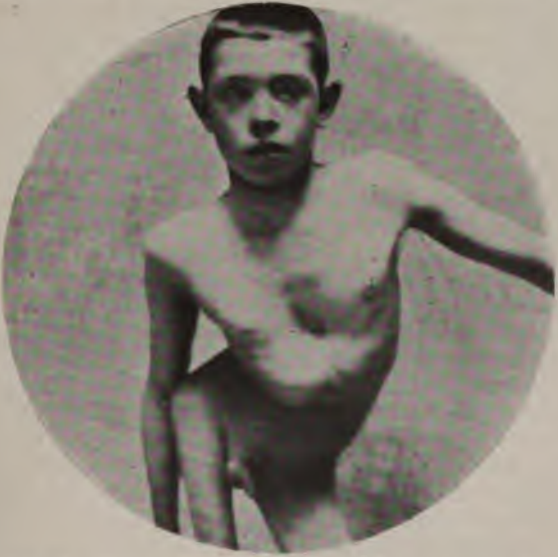

Fig. 54.—Wasting and deformities following widespread acute poliomyelitis.

of the peripheral nerves and of the anterior horns. *Peripheral neuritis* can generally be excluded by the absence of pain and of sensory disturbance, and by the irregular distribution of the paralysis and its failure to correspond to the muscle areas of peripheral nerves. Moreover, most cases of multiple neuritis of toxic origin, e.g. those due to alcohol, arsenic, and lead, develop so deliberately that there is little chance of their being mistaken for acute poliomyelitis, while that due to diphtheria, where the onset might be more confusing, usually has the typical distribution of paralysis of the soft palate and ocular muscles. Difficulty, however, occasionally arises in cases of multiple neuritis of acute onset in which no definite changes in sensation can, at any rate for a time, be demonstrated, but here again the symmetry is of great assistance.

Lesions of the muscles and peripheral nerves being excluded, the cells of the anterior horns remain to be considered. Here the only conditions that are likely to occasion confusion are those in which the function of the cells is very rapidly modified by toxins that give rise to cases included in the clinical group of *Landry's paralysis*. Here the patient rapidly loses power, as in acute poliomyelitis, but there is usually no definite wasting of muscles nor are there changes in their electrical reactions. Moreover, the loss of power in Landry's paralysis advances evenly upwards from the feet, while that of acute poliomyelitis is nearly always irregularly distributed.

While in clinical appearance and course this variety differs from the typical "infantile paralysis," it is still probable, as already stated, that the underlying virus of both Landry's paralysis and poliomyelitis is, at any rate sometimes, the same, since during epidemics the two types may be found side by side.

Prognosis.—The prognosis has to be considered (1) as regards life and (2) as regards recovery of power.

1. *Danger to life.*—From a study of epidemics it is now recognized that the danger to life is considerable. The mortality differs widely in different epidemics, in some being as low as 2 and in others as high as 20 per cent. and even higher, and it is greater in adults than in children.

Cases with bulbar symptoms are always serious, as also are those which exhibit the symptoms of Landry's paralysis, since in these latter the respiratory movements are particularly liable to be affected.

2. *Recovery of power.*—In some cases the illness consists almost entirely of general toxæmic symptoms, the local signs being absent or so slight that no anxiety arises as to any permanent paralysis. In most cases, however, there is the loss of power that is so characteristic of the disease, and in these recovery is seldom anything like complete, although there is often very considerable improvement.

The guide to the prognosis in these cases is to be found in a consideration of their morbid anatomy.

If a group of cells taken from an affected area be examined, it will be seen that some are entirely destroyed, others are badly injured, while still others on the borders of the diseased area have escaped. Those that are destroyed can never recover their function, but those in which the injury is less complete are capable of some degree of regeneration, and it is upon these latter that the ultimate degree of recovery largely depends.

As a general rule, it may be said that if all goes well some considerable degree of improvement will take place—but as to how much, time alone can show. When the paralysis is extensive the growth of all the structures will be impaired. The limb remains stunted, wasted, and colder than its fellow, and troublesome ulcers may form in the skin. As time goes on, the muscular tissues contract and produce various kinds of talipes in the leg or corresponding deformities in the arm (Fig. 55).

Treatment. (1) **Prophylactic.**—Since the disease is known to be contagious, it is desirable to isolate the patients and to disinfect the intestinal excreta and any discharge from the nose or other mucous channels in which the virus may be contained. The length of time required for isolation to be successful is uncertain, and has been placed at periods ranging from three to eight weeks.

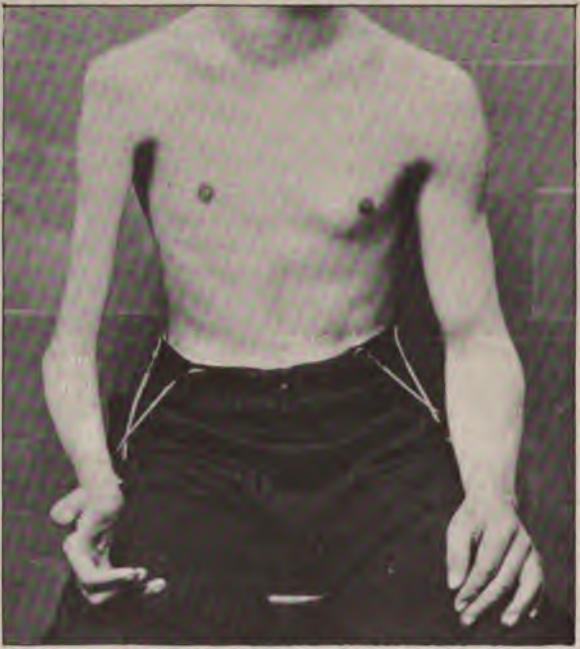

Fig. 55.—Wasting and deformity of the right hand and arm as the result of acute poliomyelitis in childhood.

Unfortunately, isolation cannot be depended upon to check the spread of the disease, the virus of which, as already mentioned, is apparently often conveyed by healthy intermediaries, and moreover in epidemics there are frequently abortive cases which remain unrecognized and serve as centres of fresh infection.

(2) **Therapeutic.**—During the febrile stage the child should be kept in bed. The bowels should be opened by a dose of grey powder or calomel, repeated if necessary, and an antifebrile mixture containing diaphoretics and diuretics may be given. Salicylates, aspirin, and phenacetin are among the drugs frequently prescribed. At this stage little more can be done, but as soon as the acute symptoms have subsided efforts must be made to maintain the nutrition of the muscles until the damaged cells recover sufficiently to resume their functions. Massage and passive movements are generally considered the most reliable methods, together with electrical treatment. Nowhere is perseverance in treatment more necessary than in this disease. The treatment must be kept up for a very long time, with the object of improving those fibres which, though badly nourished, are still, by persistent stimulation, capable of being strengthened sufficiently to aid in moving the limb. During this period every effort must be made to prevent contractures and deformities, by means of light massage, passive movements, and the judicious use of splints. Above all, the limb must never be allowed to assume such a position as to cause tension or stretching of the paralysed muscles, but must always be supported in such a manner as to keep these muscles slack. Sir Robert Jones a long time ago drew attention to the retardation of recovery which takes place in paralysed muscles that are overstretched, and he showed that, in the case of infantile paralysis, any statement concerning possibilities of recovery must, to be accurate, be based upon an estimate as to how much of the muscle paralysis is due to overstretching and how much to complete destruction of the corresponding cells in the spinal cord.

(3) **Operative.**—When the limits of improvement seem to be reached, or when there are signs of deformity, the orthopædic surgeon may be consulted with a view to improving the usefulness of the limb by operation.

CHAPTER XX

LANDRY'S PARALYSIS

This disease, first described by Landry, is characterized clinically by a paralysis which begins in the feet and rapidly extends upwards to the trunk, arms, and bulb; occasionally the parts may be invaded in the opposite order. Both sexes may be attacked, and most of the cases have occurred between the ages of 20 and 45.

Etiology.—There can be little doubt that the symptoms are the result of poisons affecting the functions of the spinal cord, and sometimes they have appeared to be the direct sequelæ of attacks of septicæmia, typhoid, influenza, and other specific fevers. There is, however, reason for thinking that in some cases the underlying toxin is similar to, if not identical with, that of acute poliomyelitis, since the symptoms of both diseases are met with in epidemics of acute poliomyelitis (*see* p. 194).

Pathology.—No definite changes in the cord can be seen with the naked eye. Microscopically, hyperæmia and swelling of the cells have been observed. Changes in the peripheral nerves have also been described. The loss of function seems to be in considerable advance of any definite changes in structure.

Symptoms.—Weakness begins in the legs, usually in one a few hours before the other, and in a very short time there is complete flaccid paralysis. The loss of power spreads upwards to the muscles of the abdomen, thorax, arms, neck, and face.

The muscles are flaccid, but usually show no definite changes in their electrical reactions and little or no signs of wasting. The tendon reflexes are lost, but the power of the sphincters is generally retained. As soon as the paralysis spreads into the cervical region the patient is in imminent danger of death from paralysis of the diaphragm; and if he escapes this danger, another is frequently met with from failure of the muscles of deglutition, with a consequent tendency for food to find its way into the larynx. Sensation is but little, if at all, altered, and the mind remains clear. The temperature may be raised, but pyrexia is not a prominent feature. The spleen is generally enlarged.

Variations.—Inasmuch as the nature of the poison is probably not the same in all cases, and the anatomical changes vary, it is not surprising that all cases do not adhere rigidly to the one type. In some the paralysis appears first at the upper end of the body and spreads downwards; in others, especially those which last a long time, there may be signs of wasting of the muscles and changes in their electrical reactions; while in others again the action of the sphincters may be impaired.

Nevertheless, there is clinically a very definite set of cases characterized by a paralysis which ascends and which is unaccompanied by wasting or by sensory disturbances.

The most practical view is to suppose that certain poisons, under certain conditions, lower the functions of the cells of the anterior horns (and perhaps other parts of the nervous system as well) sufficiently to produce a flaccid paralysis, but not sufficiently to cause wasting and electrical changes; that here and there the poison is intense enough to lead to such changes, and that gross anatomical lesions sometimes further intensify the symptoms. In this way we can understand the variation of the symptoms in different cases.

Diagnosis.—The definite march of the paralysis, and the absence of wasting, of electrical changes, and of altered sensation generally, enable the disease to be distinguished, though, from what has already been said, it will be plain that the less typical cases may occasion difficulty.

From *peripheral neuritis* Landry's paralysis differs in the extension of the paralysis to the trunk before the arms are affected, and also in the absence of wasting and of electrical changes, which are generally soon seen in peripheral neuritis.

In an *ascending myelitis*, where there is a definite inflammation spreading up the cord, there will be loss of control over the sphincters, and bedsores will soon occur.

An *infantile paralysis* (acute anterior poliomyelitis) in which the symptoms are widespread may be difficult to differentiate for a time, since both this and Landry's paralysis depend upon lesions of the motor cells of the anterior horns. As already stated, some cases are apparently only different clinical manifestations of the same infection. Usually the more scattered paralysis of the poliomyelitis makes the diagnosis clear, but where there is a difficulty it may be necessary to wait until wasting and electrical changes appear in the muscles. When a patient recovers from Landry's disease the power returns more rapidly than in recovery from infantile paralysis.

Prognosis.—A patient suffering from Landry's disease is always in great danger until the acute symptoms have subsided and the risk of respiratory failure has passed.

In a large number of cases the disease ends fatally in a few days, while in some the symptoms subside and power gradually but steadily returns.

Treatment.—In the absence of any specific antidote to the poison, reliance has to be placed on maintaining the patient's strength, and for this purpose hypodermic injections of strychnine are probably the best. Ergot has been recommended.

The breathing of the patient should be carefully watched, and an attempt be made to tide over any temporary difficulty by artificial respiration.

CHAPTER XXI

MYELITIS

Etiology.—The nature of the lesions which are included under the clinical term "myelitis" differs. In some cases there is undoubtedly a primary inflammation brought about by the direct invasion of bacilli or their toxins through the blood- or the lymph-stream; in others the underlying condition is one of thrombosis, to which the changes in the cord are secondary.

It will therefore be evident that infective diseases and syphilitic endarteritis are the most common causes. Cold, injury, and debility from any cause may, by lowering the resistance, dispose to an attack.

The most vulnerable part of the cord appears to be the lower dorsal region, and it is there that the lesion most frequently occurs.

Pathology.—The substance of the cord is softened, and in some cases the different structures are indistinguishable from one another. Hæmorrhages, engorgement of vessels, exudation, and cell degeneration are all present in varying degree. In the more chronic cases the distribution differs according as the infection arrives by way of the blood or of the lymphatics (*see* p. 72).

Symptoms.—The symptoms vary with the level and the extent of the lesion.

1. **Transverse dorsal myelitis.**—When the onset is acute, and the patient has hitherto been well, the first signs are generally a feeling of increasing heaviness in the limbs, with perhaps some difficulty in passing urine.

The numbness and weakness rapidly increase, and in a severe case the lower limbs may become within a short time both senseless and motionless. Examination shows *loss of power* below the level of the lesion, and, assuming the attack to be severe, there will be absence of all movements of the feet, knees, and hips, the limbs lying flaccid and helpless. At this early stage the *tendon reflexes* may possibly be temporarily inhibited, but they soon become increased, as in other upper motor neurone lesions. At the same time that impulses are prevented from flowing down the motor tracts, the transverse lesion also prevents stimuli from passing up the sensory columns; hence in severe cases there is *loss of sensation* of every kind below the level of the lesion, but at that level, and just above it, there is often a *zone of hyperæsthesia,* due to irritation of the sensory roots as they pass out from those segments of the cord. This zone of hyperæsthesia and the upper limit of the anæsthesia are valuable guides to the position of the lesion.

Total loss of sensibility is often accompanied by the formation of *bedsores* or crops of *herpes,* both of which are due to those disturbances in nutrition generally described as "trophic." No impulses from the bladder and rectum can reach the brain, nor can any pass downwards, with the result that control over the *sphincters* is lost. The bladder either discharges its contents reflexly at irregular intervals, or, as more frequently happens, it becomes distended until the incontinence of overflow ensues.

If the patient lives, the more acute symptoms pass off, and the permanent effects of degeneration subsequent to the transverse lesion become apparent. (Plate 10.) The fibres of the crossed pyramidal tracts degenerate downwards, with all the accompanying attributes of lateral sclerosis. The legs become *rigid,* with or without some return of power according to the intensity of the initial lesion, and the rigidity may be succeeded by *contractures,* in which, in bad cases, the strength

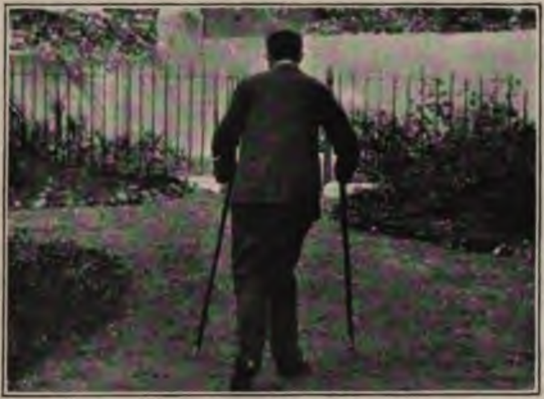

PLATE 10. — Instantaneous photograph showing gait in paraplegia following myelitis.

of the flexors and adductors overpowers that of the extensors and abductors. The *tendon reflexes* are very exaggerated, *clonus* appears, and the *plantar reflex* gives a typical extensor response. There is no wasting, and no electrical changes are present. Whether control over the sphincters is entirely or only partially regained depends upon the amount of communication that is re-established after the acute symptoms have subsided.

Such are the main points to be observed in a transverse myelitis of the lower dorsal region.

2. A **transverse lumbar lesion** destroys many motor cells of the anterior horns, from which fibres go to supply the muscles of the legs, and hence there is a considerable amount of wasting. The centres for the bladder and the reflexes are also liable to destruction; consequently the superficial and deep reflexes of the legs disappear, and the bladder becomes a flaccid bag from which the urine continually runs, i.e. true incontinence as opposed to the false incontinence of the distended bladder. As in transverse myelitis of the dorsal region, there will be anæsthesia below the level of the lesion.

3. In myelitis of the **cervical region** there will be paralysis and anæsthesia below the level of the lesion, as in the dorsal region. In addition, there is the possible presence of hyperpyrexia and of inequality of the pupils (due to interference with the cervical sympathetic nerves). If the lesion chances to be at the level of the cervical enlargement, there will be wasting of the muscles of the arm from destruction of their motor cells, just as the muscles of the leg waste when the lumbar enlargement is affected. If, when no intercostal action is possible, the motor cells of the phrenic nerves at the level of the 3rd, 4th, and 5th cervical roots are destroyed, death takes place from failure of respiration.

4. **Acute ascending myelitis,** in which the inflammatory process commences in the lower parts of the cord and extends upwards, sometimes occurs. The chief symptoms are rapidly ascending paralysis with loss of sensation,

loss of control over the sphincters, and the formation of bedsores. The whole thickness of the cord is acutely inflamed, and its structure destroyed.

5. **Disseminated acute myelitis,** in which the inflamed areas are scattered throughout the cord, is sometimes found.

Diagnosis.—In making a diagnosis it is first necessary to be certain that the lesion is in the cord, and the methods of ascertaining this point will be found described under the heading of Paraplegia (p. 183). When this is established it is necessary to consider the nature of the lesion. The acute onset suggests an inflammatory origin. Hæmorrhage into the substance of the cord, besides being very rare, has a more sudden onset, and would be unlikely to assume a transverse distribution, though bleeding into a cavity of syringomyelia might in certain circumstances give rise to difficulty.

The back must always be carefully examined to see if the symptoms may possibly be due to pressure.

Meningitis may simulate myelitis, but in the former there are usually radiating pains due to irritation of the nerve roots; the two conditions may, however, coexist, as so often happens in syphilitic disease of the cord.

An acute myelitis which ascends the cord will closely resemble a case of *Landry's paralysis*, but in the latter there are usually no loss of sensation, no loss of control over the sphincters, no wasting, and no electrical changes in the muscles.

The Wassermann reaction of the cerebro-spinal fluid will assist in determining the nature of the lesion.

When the patient is seen for the first time after the acute stage has passed, the resulting degeneration will be in evidence.

The diagnosis will then chiefly have to be made from *ataxic paraplegia, subacute combined degeneration, disseminated sclerosis,* and *tumour of the cord*. The decision must depend largely on the history of the onset and the course of the disease, since the physical signs will often be compatible with diseases of different origin.

Prognosis.—Myelitis of the cervical region of the cord always threatens to destroy life by its proximity to the nuclear origin of the phrenic nerves and to the structures of the medulla. When the disease is situated in the dorsal and lumbar region there is less immediate danger, but if the structures of the cord are permanently destroyed the exhaustion accompanying bedsores and cystitis gradually carries off the patient. Where partial recovery takes place the patient may, with care, live many years.

At the beginning of a case it is impossible to predict with any certainty how much power will be regained. Some patients recover in an extraordinary way, but the majority, after reaching a certain point, make no more progress, and for the rest of their lives suffer from various degrees of spastic paraplegia, with or without some difficulty in exercising efficient control over the sphincters.

Treatment.—It is generally recommended that the patient should lie on his side or stomach, to avoid congestion of the diseased part, but it is doubtful if position really makes any difference. Attention must be paid to the state of the bladder, and the urine should be drawn off regularly, during which operation the strictest aseptic precautions must be taken to prevent cystitis. He should lie on a water-bed, and the greatest care must be exercised to prevent the occurrence of bedsores. The bowels should be kept freely open. When there is reason to suspect syphilis as the cause, appropriate treatment by salvarsan or mercury should be given, and the latter drug also often appears to be useful in cases due to other causes. Counter-irritation is sometimes applied to the spine, but, as it is of doubtful value and may lead to the formation of sores, it is better omitted. When the acute symptoms subside, strychnine is useful, but it should not be given if much rigidity is present. Massage, passive movements, good food, and general tonic treatment are indicated at this stage. Every effort must be made to prevent the limbs from assuming undesirable positions and undergoing contractures.

extensor plantar response often remains. Œdema of the legs, bedsores, and cystitis may now occur, fever is present, and the patient loses strength and usually dies within a few weeks.

Pathology.—The cord shows degeneration chiefly of the lateral and posterior columns, which is secondary to a more localized and diffused transverse lesion generally most marked in the thoracic region of the cord.

Diagnosis.—The difficulties in diagnosis will vary according to the stages at which the disease is seen.

When the signs are those of spastic weakness and ataxy the likelihood of *disseminated sclerosis* being present has to be considered. The main points in which disseminated sclerosis differs are in the earlier age of onset, the variableness of symptoms, the tendency to optic atrophy and cranial nerve palsies, and the presence of nystagmus.

As the disease progresses the degeneration may be referred to the results of a previous *transverse myelitis* or accounted for by the presence of a *chronic myelitis*.

When the knee-jerks are lost and there is flaccid paralysis, *peripheral neuritis* has to be thought of, but the anæsthesia of the trunk, the incontinence of urine, and an extensor plantar response are against it, as also is the history of the disease.

The loss of power, wasting and electrical changes in the muscles, together with absence of the Argyll-Robertson pupil, will enable *tabes* to be excluded.

Sometimes the signs may suggest the presence of a *tumour*, but the pains are unlike those due to pressure on roots, and the paralysis and sensory changes do not show the signs of being due to pressure on one half of the cord more than the other, as often happens in the case of tumours.

In the earlier stages subacute combined degeneration may have to be diagnosed from *hysteria*. In doubtful cases the most reliable sign of organic disease is to be found in the extensor response of the plantar reflexes. If this is obtained the organic nature of the case is clear.

Where there is any uncertainty, these reflexes should be repeatedly examined, since they do not always respond in the same manner.

Prognosis and treatment.—Hitherto all the severe cases recorded seem to have ended in death, nor has their course been materially modified by any drugs. Treatment must be directed towards improving the general health, which is always bad. Careful search should be instituted for any source of infection; if this is found and successfully treated, the symptoms may be ameliorated, or even arrested.

CHAPTER XXIII

DISSEMINATED SCLEROSIS

Etiology.—Very little is definitely known about the origin of disseminated sclerosis.

While there is no exact age limit, the symptoms begin in most cases between the ages of 15 and 35. Children occasionally suffer, but elderly people are very rarely attacked.

The number of cases occurring in the two sexes is about equal.

Pathology.—The disease, as its name implies, is characterized by the formation of islets of connective tissue which are scattered throughout the length of the central nervous system and also to some extent in the substance of the peripheral nerves, more especially in those of cranial origin. The disease in classification therefore belongs both to the brain and the cord.

The white matter of the brain and cord is principally affected, it being somewhat less common to meet with symptoms attributable to alterations in the grey substance.

The patches, which are greyish-white in colour and usually sharply defined, vary very much in size, some being as large as peas and discernible with the naked eye, while others are so small that they are only recognizable with a microscope.

The changes appear to begin in the neuroglia rather than in the nerves fibres, though on this point there is some difference of opinion. In any case the axis cylinders are the last structures to degenerate, which accounts for the fact that there is seldom any secondary degeneration to be detected either above or below the sclerosed site.

As the symptoms in the earlier stages of the disease are liable to remissions, it is probable that the patches are also liable to variations before they become permanently established. These early changes may be due to vascular disturbances with possibly some passing œdema, and their variations probably correspond to the remissions and exacerbations which clinically are so characteristic of the disease.

The principal suggestions which have been put forward to account for the origin of the patches are :

1. That they are due to the effect of toxins.
2. That they are the result of some congenital abnormality of the nerve tissue.
3. That they are due to a micro-organism.

In favour of the first view, it is pointed out that the symptoms sometimes appear to arise after the infection of specific fevers. But where a specific fever has preceded the symptoms, the probability is that it has only aggravated them, or perhaps precipitated their occurrence. Congenital changes, probably beginning with a proliferation of neuroglial tissue after the manner of that which takes place in syringomyelia, have been suggested as the cause, but there seems to be no definite evidence to support this hypothesis.

Of late years the view that the disease depends upon some infective organism has been largely held. Several observers claim to have found a spirochæte in the cerebrospinal fluid of some cases, and there is little doubt that this view best explains the variations and remissions that so often occur during the course of the disease.

Symptoms.—In the form which has generally been considered characteristic of the disease the symptoms are mainly *weakness*, *spasticity*, and *ataxy* of the lower limbs, together with alterations in *speech*, *tremors*, *nystagmus*, and *optic atrophy*. (Fig. 56.) This combination of signs makes up a very striking picture.

The patient walks with some degree of difficulty; the feet shuffle on the ground owing to the rigidity which impairs the freedom of the joint-movements, while

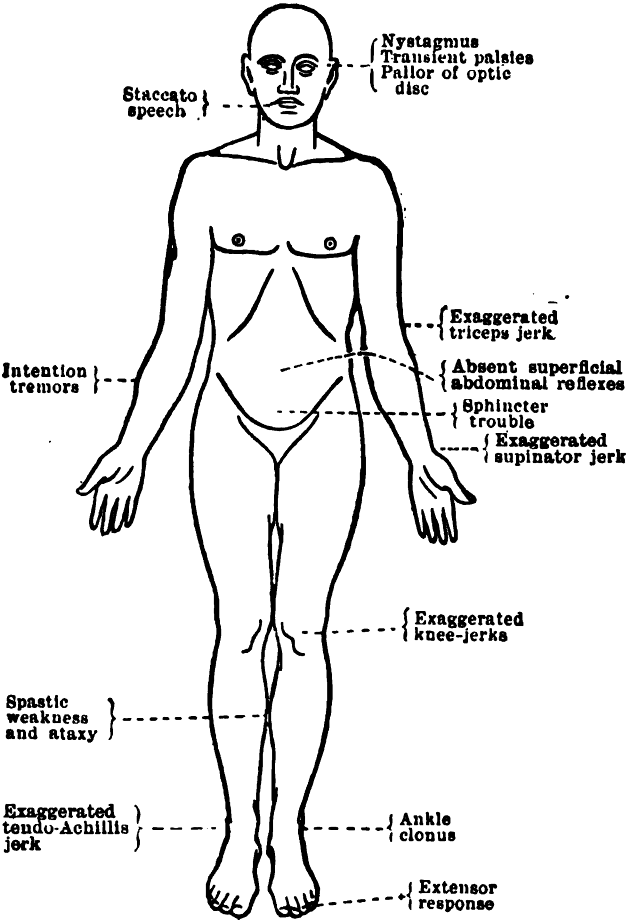

Fig. 56.—Scheme of the principal symptoms occurring in the course of disseminated sclerosis.

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l
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there is also an unsteadiness of gait owing to changes in the posterior and cerebellar tracts.

When the patient is seated there is little to notice, yet any movement of the arm or hand is accompanied by a coarse tremor, which increases in amplitude as the movement is continued. Thus, if the patient be asked to carry a glass of water to his lips, the movements become more and more exaggerated, until the contents of the glass are dashed to the ground. (Plate 11.) In the same way, if he be asked to write, the pen is drawn spasmodically across the paper, though, of course, in the earlier stages, all grades of impairment of writing are encountered. (Fig. 57.) In those engaged in clerical work this symptom is sometimes the first noticed. This tremor, often known as "volitional" or "intentional" tremor, is by no means necessarily confined to the arms. Sometimes there are rhythmical movements of the head, which, however, cease when the muscles of the neck are supported on a pillow.

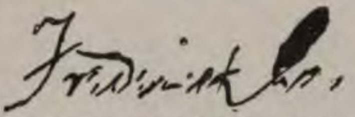

Fig. 57.—Specimen of writing in a case of disseminated sclerosis.

The tremor of disseminated sclerosis, then, is characterized by—

(1) Its occurrence during voluntary movements.
(2) Its cessation during rest.
(3) The coarseness of its vibrations, which increase in amplitude with the continuation of the movement.

The power of the legs (and probably of the arms) will be found to be considerably less than normal.

The knee-jerk and tendo-Achillis jerk are increased, ankle-clonus is often obtained, and the plantar reflex shows the extensor response; there may also be some tendency to clubbing of the feet.

In the upper limb also the deep reflexes are generally found to be exaggerated, and a brisk jaw-jerk will in all probability be obtained, but the superficial reflexes of the abdomen are frequently lost.

As regards the ocular signs, nystagmus is observed when the patient attempts to look to one or the other side, and there may be some weakness (usually transient) of one or more muscles, giving rise to double vision. The pupils usually react normally to light and on accommodation. Changes in the optic nerves occur in a large proportion of cases of disseminated sclerosis, and failure of vision may be the patient's earliest complaint. The alteration most commonly seen is that of pallor of the optic disc, indicating a certain degree of atrophy. In a very small percentage of cases signs of antecedent neuritis have been noted. The atrophy is often confined to the temporal half of the disc, and frequently affects only one eye. Only exceptionally, therefore, is total blindness likely to occur. The fields of vision may show restricted perception of colours before the sensation to ordinary light is demonstrably impaired.

The speech is known as "scanning" or "staccato," from the pronounced manner in which every syllable is jerked out.

There is generally impairment of sphincter control at some time during the course of the disease.

Mental instability is often apparent, as is shown by a tendency to laugh and cry when there is but slight emotional stimulus; occasionally epileptiform fits occur.

Such are the main points to be observed in a so-called typical case, but it by no means follows that the signs always appear in this pronounced form; indeed, the majority of patients when first seen do not present a picture anything like this, and may go on for years with vague and transient symptoms, the cause of which is often attributed to hysteria unless careful examinations are repeatedly made. It therefore happens that these so-called atypical cases are of the greatest importance, because (1) they are more numerous than those in which signs are from the first permanent and pronounced, (2) they are so apt to be diagnosed as functional, and (3) any advantage that can be obtained from treatment is likely to be far greater when the changes in the nervous tissue are capable of variation than when they have become permanent.

The patient, very commonly a girl, and usually somewhat above the age of puberty, may complain of peculiar sensations of numbness which come and go in a mysterious manner, and for which no obvious cause can be found, although they sometimes tend to be more severe after fatigue or at the time of the menstrual period. Examination during such an attack may or may not reveal a patch of anæsthesia which does not seem to correspond to any definite anatomical arrangement, and then perhaps everything passes off for a time, and all is forgotten until, later on, one of the legs drags, or all power suddenly goes out of the arm, again to return or vary from time to time. It may be (and this is very significant) that there is a temporary complaint of double vision, and there is very often transient difficulty in exercising full control over the sphincters.

In all such cases the greatest care must be taken before organic disease can be excluded, and where the history is suspicious and the results of examination are negative it is often wise, if possible, to postpone giving a definite opinion, for signs which are absent at one time may frequently be elicited with ease at another.

Pallor of an optic nerve, nystagmus, squint, slight incoördination of the arms, and an extensor response of the plantar reflexes are among the most important things to note, and the establishment of one sign which is of definitely organic origin is as useful in a difficult case as is the discovery of a single tubercle bacillus in a case of suspected pulmonary disease.

In other cases, although the symptoms are obviously of organic origin, they may for a time be so restricted as to give rise to doubt regarding their nature, as, for example, when a spastic or an ataxic paraplegia is the only condition present.

In yet others the patches of sclerosis, though widely distributed, may not be in the positions to produce the typical signs of the disease, and thus arises another set of cases not conforming to type.

Diagnosis.—It will be evident that the diagnosis of disseminated sclerosis may be very easy or the reverse, and further, that the difficulties will vary during different phases of the disease.

1. During the early stages of its development the tendency is to mistake it for *hysteria*. The chief points to be noted in making the diagnosis have already been mentioned.

2. When the patches are situated exclusively in the course of the fibres of the lateral tracts, *primary lateral sclerosis* will be thought of, and, in the absence of any further signs, time alone may determine the diagnosis; but it must be remembered that primary lateral sclerosis is rare, and that many cases which appear at first to be of this nature eventually develop signs of the disseminated type.

Aid may often be obtained from the variability of symptoms, for primary lateral sclerosis is a disease of gradual but steady progress, whereas remissions are very characteristic of disseminated sclerosis.

When there are patches in the posterior in addition to the lateral columns, and no symptoms point to sclerosis elsewhere, the diagnosis for a time may be *ataxic paraplegia*, or an *incomplete transverse myelitis*. Ataxic paraplegia is, however, a clinical manifestation that may depend upon various underlying pathological conditions, and it is therefore always wise, when making the diagnosis, to keep in mind the further developments which so many of these cases undergo.

3. When the signs are well developed, yet are not those which characterize the recognized types of the disease, there may be some hesitation in distinguishing the condition from *Friedreich's ataxy*, *paralysis agitans*,

cerebellar disease, and, in children, from *cerebral diplegia*. *General paralysis of the insane* sometimes shows symptoms which resemble those of disseminated sclerosis, but the inequality of the pupils and the loss of the light reflexes, together with the mental changes present, do not usually leave the diagnosis in doubt.

Paralysis agitans can seldom give rise to real difficulty, for the tremor is finer, occurs during rest, and is diminished rather than otherwise on movement, while rigidity is seldom absent.

Confusion with cerebellar disease is only likely to arise in very chronic cases. In cerebellar tumours optic neuritis, headache, and vomiting are distinguishing features. Cases of cerebellar ataxia of childhood may sometimes resemble disseminated sclerosis, as also may some of the less typical instances of cerebral diplegia. A tremor which closely resembles that of disseminated sclerosis may sometimes arise from *chronic alcoholism* and also as a result of *chronic mercurial poisoning* (Fig. 58), but the history of these cases, and the ages at which they occur, together with the absence of other signs of disseminated sclerosis, generally make the diagnosis clear.

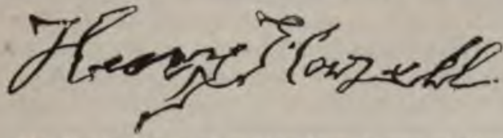

Fig. 58.—Specimen of writing from a case of chronic poisoning by mercury.

Prognosis and treatment.—The outlook for patients with disseminated sclerosis is not hopeful, since experience has shown that, despite the transient nature of certain early symptoms, a temporary improvement is the most that can generally be expected. Sometimes, however, there are remissions even for years; and, very occasionally, cases have been recorded in which no relapse seems to have taken place. When the patient comes under treatment early, it is doubtless possible frequently to retard the course of the disease by insisting on the avoidance of fatigue during the stages of remission, and on complete rest during those of exacerbation.

Vaccines and inoculations of different kinds have been recommended with little success, but it is possible that these forms of treatment will be further developed in the future if, as seems likely, it becomes definitely established that a micro-organism is the cause of the disease. Urotropin may be tried on account of its supposed antiseptic effects on the cerebro-spinal fluid.

The general nutrition should be carefully maintained by a diet containing an abundance of milk. Tonics, especially those containing iron, are useful; arsenic also has gained some reputation in this disorder, but no drug is known to have any specific effect upon the lesions. Massage, carefully regulated, is of use during certain phases, and in the more chronic conditions selected exercises may be tried with a view of improving the accuracy of the movements.

CHAPTER XXIV

PROGRESSIVE MUSCULAR ATROPHY AND PROGRESSIVE BULBAR PARALYSIS

PROGRESSIVE muscular atrophy is the title given to those cases in which wasting of muscles results from a slow and progressive degeneration of the motor cells in the anterior horns of the spinal cord, or of their analogues in the medulla and midbrain.

In order, however, adequately to understand the nature of the disease, it is necessary to take a rather wider view of its pathology and to regard it as an affection of the whole motor system (i.e. of the cell bodies of both upper and lower neurones), in which the lower neurones are generally the more severely attacked.

Etiology.—Most of the cases occur towards middle life; more men suffer than women, and heavy manual labour sometimes appears to be connected with the onset of the disease. In a certain proportion of the cases the degeneration appears to be due to parenchymatous syphilis. In others the cause is uncertain, and all that can be stated is that some poison, the nature of which is at present unknown, causes a progressive degeneration to take place in the cell bodies of the motor neurones.

Symptoms.—The usual clinical picture of progressive muscular atrophy is due to the predominating degeneration of the cells of the lower motor neurones, those of the upper neurones being generally affected to a less degree. The degeneration of these lower neurones does not always begin at the same level. Most often the cells of the cervical region of the cord are the first affected, and next in frequency those of the medulla. Less often the cells of the lumbar region are attacked first, and least often of all the first signs of the disease arise from degeneration of the nuclei of the ocular nerves. (Fig. 59.)

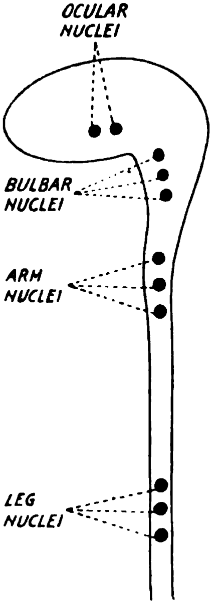

Fig. 59.—Diagram showing different positions in which progressive muscular atrophy may commence.

When the disease begins, as it generally does, in the cervical region, the cells at either the upper or the lower end of the cervical enlargement usually degenerate first, and consequently the wasting is first noticed in the muscles of the shoulder or of the hand. In the hand there is wasting of the interossei and of the muscles of the thenar and hypothenar eminences (Fig. 60). Depressions appear between the metacarpals, and the outline of the first of these bones becomes unduly prominent as the fibres of the first dorsal interosseous muscle (i.e. the abductor indicis) shrivel. The fingers can no longer be fully separated, and the power to abduct and oppose the thumb is lost. At the same time the hand gradually assumes a claw-like shape (*main-en-griffe*), and the thumb tends to rotate so that it looks like a fifth finger, and thus resembles the thumb of an ape. The reaction of the wasted muscles to faradism and galvanism is diminished, and later may altogether disappear, but there are generally some fibres left which still show some reaction to faradism right up to the end, and hence the "reaction of degeneration" is not usually present.

The reflexes in the diseased areas disappear as their arcs become broken by the degeneration of the motor cells.

As the motor cells are alone affected, there is no loss of sensation, nor is there usually any pain beyond that of aching and stiffness, which may well be due to the changes taking place in the muscles. Fibrillary tremors are very often present.

After a time, perhaps a few months, wasting begins in the opposite hand, and as degeneration extends to other levels of the cord the muscles of the shoulder and trunk waste, and the weakness of the trunk muscles may imperil the adequacy of the respiratory movements, thus disposing to pulmonary complications.

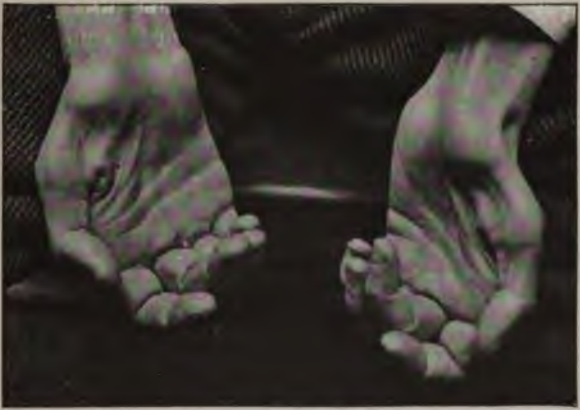

Fig. 60.—Wasting of hands in progressive muscular atrophy.

In other cases the disease spreads upwards to the nuclei of the bulb, giving rise to additional symptoms of **progressive bulbar paralysis,** which is practically a progressive muscular atrophy in which the nuclei of the bulb are attacked.

In the bulb the cells chiefly affected are those of the 10th, 11th, and 12th nuclei; hence there is loss of power in the lips, tongue, pharynx, and larynx, as described in one of the titles of the disease—*progressive labio-glosso-pharyngeal and laryngeal paralysis.*

The degeneration of the hypoglossal nucleus accounts for the tongue symptoms, the affected accessory portion of the 11th and vagal nuclei causing the pharyngeal and laryngeal troubles. The one difficulty is to account for the affection of the lips without assuming degeneration of the facial nucleus, of which there is often no evidence, and the solution appears to be that those fibres which supply the orbicularis oris, though running in the facial trunk, in reality have their origin from the upper end of the hypoglossal nucleus, and therefore share in the degenerative changes of the latter.

In a typical example the gradual degenerative changes in the hypoglossal nucleus first show themselves by some weakness in the more delicate movements of the tongue, so that certain letters which require a firm muscle for their articulation cannot be clearly pronounced. Some such letters are *l*, *n*, *r*, *s*, *t*. As the change progresses, the tongue wastes and becomes more and more powerless, until at last it cannot be pushed out or thrust into either cheek, and finally it lies helpless on the floor of the mouth with its loosely fitting mucous membrane wrinkled over it. At the same time the lips, formed by the orbicularis oris, which derives its nerve supply from the upper part of the hypoglossal nucleus, also show signs of weakness and wasting. At first, certain letters, such as *o*, *u*, *p*, *b*, *m*, requiring the lips to be pressed closely together for their formation, are blurred, and later the lips become thin, wasted, and so weak that they can no longer be approximated, the saliva constantly dribbling from the mouth. It is the drooping of the lower lip that helps so much to give the characteristic facial expression to these cases.

The degeneration of the 10th and 11th nuclei causes various degrees of difficulty in swallowing. Fluids regurgitate through the nose, and the voice becomes "nasal" owing to deficient movement of the soft palate incompletely separating the oral from the nasal cavity, while the loss of the pharyngeal reflex is a source of danger by allowing the food to pass into the larynx.

When the disease begins in the lumbar region, symptoms similar to those described for the arms are first noticed in the feet and legs; and in the rarer instances where the nuclei of the 3rd, 4th, and 6th cranial nerves degenerate, a loss in the corresponding ocular movements will be seen.

So far, we have considered the effects of the changes in the cells of the lower motor neurones. But while the degeneration of the cells in the anterior horns of the cervical region of the cord is proceeding, some degeneration is frequently also taking place in the fibres of the lateral columns of the cord, as is shown clinically by increased knee-jerks, extensor response of the plantar reflex, and some degree of weakness and spasticity of the lower extremities.

This degeneration of the lateral columns is secondary to changes in the motor cells of the upper neurone (i.e. in the cells of the motor area of the cortex), to which allusion has already been made. The effects of such upper neurone changes are, of course, masked at levels where advanced lower neurone degenerations are present, but as in the majority of cases of progressive muscular atrophy the lower neurone changes are for a considerable time limited to the arms, the legs remain free to show the signs of upper neurone degeneration. Hence, in cases of progressive muscular atrophy, *wasting of the arms is commonly found combined with some degree—often very slight—of spasticity in the legs,* together with the corresponding changes in the reflexes, viz. increased knee-jerks and extensor response of the plantar reflex.

This association of symptoms leads us a step farther, for when these spastic signs in the legs are well marked the condition is known as **amyotrophic lateral sclerosis** (i.e. a progressive muscular atrophy of the arms and a lateral sclerosis of the legs). No sharp distinction can, then, be drawn between progressive muscular atrophy and amyotrophic lateral sclerosis, for in so many cases of progressive muscular atrophy there is, in addition to degeneration of the cells of the anterior horns, some degeneration of the lateral columns, and it is according to the degree of this degeneration that the case assumes the picture of progressive muscular atrophy or of amyotrophic lateral sclerosis. Some authors deny that there is any separate condition which should be known as amyotrophic lateral sclerosis, and argue that, since most cases of progressive muscular atrophy show some signs of lateral sclerosis, the separation is an artificial one. Others, although recognizing the frequency of the presence of some degree of lateral sclerosis in progressive muscular atrophy, prefer to reserve the title of amyotrophic lateral sclerosis for the cases in which the degeneration of the lateral columns is a prominent and progressive feature of the disease. While every gradation can doubtless be recognized, the two terms are useful clinically to indicate whether the upper neurones have or have not undergone any material degree of degeneration. (*See* p. 234.)

" Progressive bulbar paralysis," though really relating to a part of progressive muscular atrophy, must similarly be retained as a useful clinical term.

Diagnosis of spinal forms.—When the degeneration begins in the nuclei of the cord the wasting indicates at once that the disease is in the lower neurone. This narrows the issue down to lesion of the anterior horns, the peripheral nerves, and the muscles themselves.

Peripheral neuritis must be excluded by the absence of pain, of anæsthesia, and of the reaction of degeneration, and the grounds for decision will be further strengthened if the area of wasting tends to correspond to the distribution of motor roots and not to that of peripheral nerves.

Myopathies.—Some of the myopathies are difficult to distinguish from progressive muscular atrophy, especially when they commence in the muscles attached to the shoulder-girdle, but when they occur in young people about or before the age of 20, or when they attack the facial muscles round the angle of the mouth, their nature is generally clear.

Syringomyelia.—Syringomyelia closely resembles progressive muscular atrophy when it commences with wasting of the hand muscles. Its nature is demonstrated by the loss of sensation for pain and temperature, while that for touch is generally unimpaired.

When the upper neurone is also extensively degenerated the wasting in the arms is often combined with spastic paralysis of the legs, and the disease then merges into that of *amyotrophic lateral sclerosis,* from which it can never be very sharply distinguished.

Diagnosis of bulbar forms.—When the degeneration begins in the nuclei of the medulla, the principal conditions it might be confused with are those arising from diphtheria and myasthenia gravis.

In *postdiphtheritic paralysis* the symptoms are often those of bulbar distribution—e.g. difficulty in swallowing and regurgitation of fluids through the nose—but the rapid onset is unlike the progressive form. The history of an antecedent attack of diphtheria makes the diagnosis plainer, and in the absence of this the subsequent history of the postdiphtheritic paralysis soon clears the matter up. Exceptionally, the effects of the diphtheritic poison are more permanent ; thus, on seeing the patient for the first time, an erroneous diagnosis might be made, but the non-progressive character of the lesion would probably arouse suspicion as to the real nature of the condition.

In *myasthenia gravis* the symptoms vary from time to time, and moreover there is seldom any marked degree of wasting. The myasthenic reaction, if present, is diagnostic.

Vascular lesions in the bulb (especially thrombosis) will cause paralytic symptoms if they occur in the neighbourhood of the nuclei. Their acute onset and more variable course will serve as distinguishing features.

Acute inflammation of the bulbar nuclei may occur in cerebral forms of acute poliomyelitis and in cases of lethargic encephalitis (*see* p. 341).

Diagnosis of the ocular forms.—In those rare instances where the nuclei of the eye muscles are the first to degenerate, ocular palsies due to tabes, myasthenia gravis, syphilis, and cerebral tumours have to be excluded.

Prognosis.—The course which progressive muscular atrophy usually runs is sufficiently implied by its name.

Extension to the trunk or bulb is generally only a matter of a few years at the most, and after either event the patient's life must always be precarious. The dangers from bulbar paralysis are choking and bronchopneumonia due to particles of food passing into the lungs, and an attack of bronchitis is very apt to cause death when the trunk muscles of respiration are wasted.

The prognosis of the parenchymatous syphilitic forms is better than that of other varieties, since there is more hope of the degenerative processes becoming arrested. It is, therefore, important to test the Wassermann reaction in all cases.

Treatment.—In the syphilitic cases the effects of specific treatment should be tried. Where the cause is unknown, all that can be done is to endeavour to counteract its effects by keeping up the nutrition of the nerves and muscles.

With regard to drugs, strychnine, iron, cod-liver oil, and other tonics are all useful. Strychnine, given regularly, seems sometimes to check the extension of degeneration, and it is thought to be more efficient when it is given under the skin. When there is much spasticity, strychnine should not be given.

Massage and electrical treatment in moderation help to keep up the strength of the muscles.

When bulbar symptoms are present the patient must be fed carefully to prevent food from going into the air-passages. In bad cases a tube may be required.

Progressive Muscular Atrophy in Infants

A form of progressive muscular atrophy which occasionally occurs in infants is known as the Werdnig-Hoffmann type, after the authors by whom it was first described. The symptoms closely resemble those of a myopathy, but characteristics of the progressive form are usually to be found in the more rapid course which it runs, and in the tendency which it shows to attack the nuclei of the bulb.

CHAPTER XXV

SPASTIC PARAPLEGIA; AMYOTROPHIC LATERAL SCLEROSIS; ATAXIC PARAPLEGIA

SPASTIC PARAPLEGIA

PRIMARY spastic paraplegia, or lateral sclerosis, is an example of an uncomplicated lesion of the upper motor neurones, and is characterized by a degeneration of those fibres only which compose the lateral tracts.

Etiology.—This degeneration appears to be the result of nutritional changes in the motor cells in the cerebral cortex from which the fibres arise, but to what these alterations in nutrition are due is uncertain. Syphilis, injury, lead-poisoning, and the toxins of acute infective disease have all been assigned as causes, and from recent investigations it appears clear that at any rate a certain number of cases are due to neuronic degeneration following parenchymatous syphilitic lesions.

In the majority of cases the disease is first noticed between the ages of 20 and 40.

Symptoms.—The main features of this disease are progressive weakness and rigidity, which first show themselves in the lower extremities. The patient notices that he cannot walk so far as he could formerly, and that, after resting, the limbs feel stiff upon using them again; this stiffness is more especially noticed when he first gets up in the morning. As the symptoms get worse he seeks advice, and it is then that the signs of degeneration of the upper neurones are detected. *Loss of power*, *rigidity*, *increased tendon reflexes*, and the *extensor plantar response*

(Fig. 61) are present, but there is no obvious wasting, and there are no changes in the electrical reactions of the muscles. There is no loss of sensation or sphincter

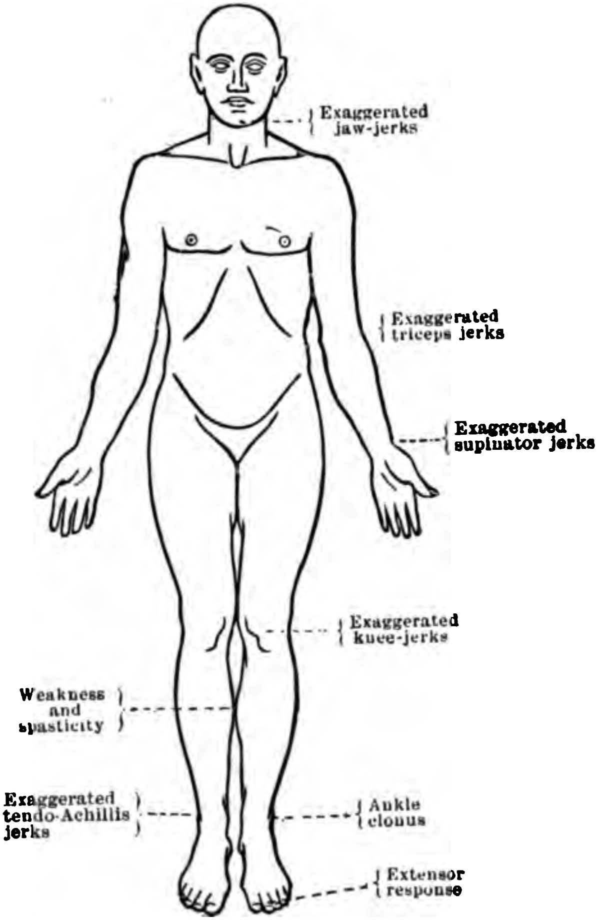

Fig. 61.—Scheme of the principal symptoms in spastic paraplegia.

trouble, and there are, in fact, no other signs to be found than those which can be accounted for by degeneration of the crossed pyramidal tracts.

As a rule the symptoms get gradually and very slowly worse, until at last walking becomes difficult or impossible. The rigidity is always a marked feature, and sometimes the joints, when moved beyond a certain distance, spring into the position of flexion or extension from which the term "clasp-knife" rigidity arises.

The *gait* is that known as *spastic*, and is chiefly dominated by the rigidity which prevents the feet from being freely raised. At the same time, owing mainly to weakness of the flexors of the ankle-joints, the toes are dragged along the ground; hence the gait is stiff, and the feet are shuffled along the ground. Cramps and spasmodic twitchings of the legs are often complained of.

Diagnosis.—The diagnosis of a case of lateral sclerosis will be conducted somewhat as follows:

The patient will probably come complaining of loss of power and stiffness, the existence of which will be confirmed. On testing the deep reflexes, the knee-jerks and the tendo-Achillis jerks will be found exaggerated, as also may be the tendon reflexes of the arm and the jaw-jerk. The plantar reflexes will be active, and the big toe will turn deliberately upwards (the extensor response).

This combination of *loss of power, spasticity, increased deep reflexes,* and an *extensor response* warrants an inference that the lateral columns are at fault, and further evidence of a negative kind in support of this inference will be found in the absence of wasting and of electrical changes in the muscles.

Signs may also be present in the arms showing that the degeneration is going on at that level. If nothing else be found, we are face to face with degeneration of the lateral columns only, and, always bearing in mind the possibility of the clinical picture changing with time into one of disseminated sclerosis, or of the revelation of some gross lesion which may for a time be indiscernible, the diagnosis of primary spastic paraplegia may be made. Primary lateral sclerosis uncomplicated by lesions of other columns is, however, a rare condition; indeed, some authors have gone so far as to doubt its existence, and incline to the belief that all cases which at first appear to be of this kind subsequently develop other symptoms which show that the degeneration is in reality more widely spread. This doubt is largely justified by the scanty pathological evidence that can be brought to show that the degeneration remains confined to the lateral columns throughout the whole course of the disease.

On the other hand, degeneration of the fibres of the lateral columns occurs frequently in association with degeneration of the motor cells of the cord, giving rise to the combination of symptoms known as amyotrophic lateral sclerosis, and therefore there appears to be no reason why in certain cases the fibres of the lateral columns should not degenerate independently of those of the lower neurone, and give rise to primary lateral sclerosis.

In any case, the greatest care must be taken to exclude the possibility of an extending lesion before concluding that the disease is likely to remain a paraplegia due only to primary degeneration of the fibres of the lateral columns.

A considerable number of cases, in which at the time of examination the only parts apparently affected are the lateral columns, prove later to have been examples of disseminated sclerosis in the early stages, as is shown by the subsequent appearance of such signs as nystagmus, scanning speech, ocular palsies, and loss of control over the sphincters.

Spastic paraplegia may be **secondary** to many conditions, among which *spinal caries, tumours, myelitis, syphilis of the cord*, and *injury of the spine* may be mentioned. In cases of doubt, the Wassermann reaction of the cerebro-spinal fluid should be tested. It may happen that in any of these the spastic symptoms are for a time sufficiently predominant to make it appear that the lateral columns alone are diseased. When, for instance, in spinal caries, the cord is compressed by inflammatory and granulation tissue, there are often, for a long time, no well-marked signs of impaired conduction of sensory impulses, although the posterior columns are presumably affected. The functions of motor fibres are the more easily disturbed, and thus signs of disease of the lateral columns appear first, and, unless the back be carefully examined, the case may be passed as one of primary spastic paraplegia.

Implication of the posterior columns (*ataxic paraplegia*) will be recognized by the presence of ataxy and the other signs of posterior column disease (*see* p. 237).

Lastly, spastic paraplegia may be closely simulated by *hysterical paraplegia*.

In organic disease almost every kind of symptom may in its turn be absent from any particular case, but when present such symptoms do not contradict one another, as they so frequently do in hysteria. In lateral sclerosis the signs that are present should be compatible with each other. For instance, the degree of sclerosis which gives rise to an increase of the knee-jerk should also give an extensor reaction of the plantar reflex, and therefore when, in paraplegia, an exaggerated knee-jerk occurs in company with a permanent flexor response of the plantar reflex, it is a contradiction of signs which should at any rate arouse suspicion. Ankle-clonus is useful, when it is well obtained, as a sign of organic disease. In functional disease the clonus, when it occurs, is generally feebly sustained, and ceases when the foot is well pressed home. It must, however, be remembered that this type of clonus, known as pseudoclonus, often occurs during different phases of organic disease.

In the gait of functional disease the whole leg is dragged after the patient in a characteristic manner, and there are generally other signs, such as anæsthesia, and variations in symptoms which are not compatible with primary lateral sclerosis. Further, a sudden onset is against primary lateral sclerosis, which usually comes on quite gradually.

Pathology.—The characteristic changes are those produced by a degeneration of the crossed pyramidal tracts, but it is rare to find a spinal cord in which these alone are affected. The degeneration probably begins in the nerve fibres themselves, and the overgrowth of neuroglia is secondary. Probably the cause, as already stated, is failing nutrition of the cortical cells, on which the nutrition of the fibres depends. There is ground for believing that the degeneration is in many cases due to parenchymatous syphilis.

Prognosis.—Degeneration limited to the lateral columns is not a direct danger to life. The changes progress slowly, and last over an indefinite number of years. Sometimes the degeneration appears to be arrested, but there is no possibility of cure in the sense of restoration to the previous condition of health.

Treatment.—No methods of ensuring arrest of the primary form are known, but general tonics must be given. Strychnine is often unsuitable because it increases the rigidity.

The general principles of treating rigidity (p. 41) are applicable here.

In many of the secondary forms the possibility of removing the cause of the degeneration is, of course, much greater.

Where the condition appears to have a syphilitic origin, anti-syphilitic treatment should be given; but if the degeneration depends upon the parenchymatous form, no striking results are likely to be obtained.

Where the trouble appears to be due to a tumour the desirability of calling in the aid of surgery must be considered.

In *caries of the spine* (Pott's disease) the pressure is usually produced, not by the displaced vertebræ, but by the inflammatory and granulation tissue which forms round the cord. This adventitious tissue may in favourable circumstances be absorbed, and treatment by absolute rest and fixation of the spine over a prolonged period (it may be for one or two years or even more) very frequently results in recovery of the nervous symptoms and arrest of the disease. In more unfavourable cases an operation such as transversectomy for evacuating an abscess or laminectomy for relief of pressure may be necessary, but the results of laminectomy in these cases are not satisfactory, and it should only be performed when the circumstances seem specially to require it.

Fresh air, cod-liver oil, iodide of iron, and other methods of improving general nutrition are all helpful.

AMYOTROPHIC LATERAL SCLEROSIS

In amyotrophic lateral sclerosis, degeneration of both upper and lower motor neurones takes place at the same time. Clinically, the symptoms are a combination of those of lateral sclerosis and progressive muscular atrophy, and reference has already been made to the relations between amyotrophic lateral sclerosis and the last-named disease (p. 223).

Etiology.—In some cases the degeneration is due to parenchymatous syphilis; in others no definite cause is known.

Pathology.—The essential cause of the degeneration is failure of the nutrition of the motor cells of the cerebral cortex and of the anterior horns of the cord. Sections of the cord show sclerosis of the pyramidal tracts and wasting of the motor cells of the anterior horns, while in the brain some degeneration of the motor cells of the cortex can be demonstrated.

Symptoms.—The symptoms vary according to the extent of the wasting and spasticity, but in a typical case the picture is one of lateral sclerosis together with wasting of the muscles of the upper limb. The onset in such a case is insidious, and usually resembles that of primary lateral sclerosis, in which weakness and stiffness of the legs are the early signs; but in some cases wasting of the hands is the first sign to attract attention, and then the onset more closely resembles that of progressive muscular atrophy.

The signs of sclerosis of the pyramidal tracts are generally very obvious in amyotrophic lateral sclerosis as shown by the exaggeration of all the tendon reflexes, the occurrence of the plantar extensor response, and the presence of clonus, which latter can often be obtained in the arms and jaws as well as in the legs. (Fig. 62.) At the same time some wasting can be noticed to be taking place in the muscles of the hands or arms.

In other cases the wasting is, at any rate for a time, the predominant feature; the rigidity and weakness are slight, and the clinical picture is then more nearly like that of progressive muscular atrophy than that of lateral sclerosis.

Diagnosis.—The diagnosis of amyotrophic lateral sclerosis is made from evidence of the coexistence of sclerosis of the lateral columns and degeneration of cells of the anterior horns of the cord. It thus forms a link between *primary lateral sclerosis* and *progressive muscular atrophy*—and, indeed, there are few cases of progressive muscular atrophy in which some signs of degeneration of the lateral columns cannot be found. Whether all such cases should be included in the category of amyotrophic lateral sclerosis is largely a matter of individual opinion. It is on the whole, perhaps, best to speak of such cases as progressive muscular atrophy associated with signs of commencing degeneration of the upper motor neurone, and to reserve the term amyotrophic lateral sclerosis for those in which the spasticity is a predominant feature from the beginning; but no sharp line of demarcation can be drawn, since cases of every gradation are met with. When the symptoms arise insidiously the nature of the disease may for a time be overlooked, and the association of the flabby muscles of the arms with stiffness and weakness of the hands may be attributed to *rheumatoid arthritis.* In considering the diagnosis, the possibility of syphilitic origin should not be overlooked.

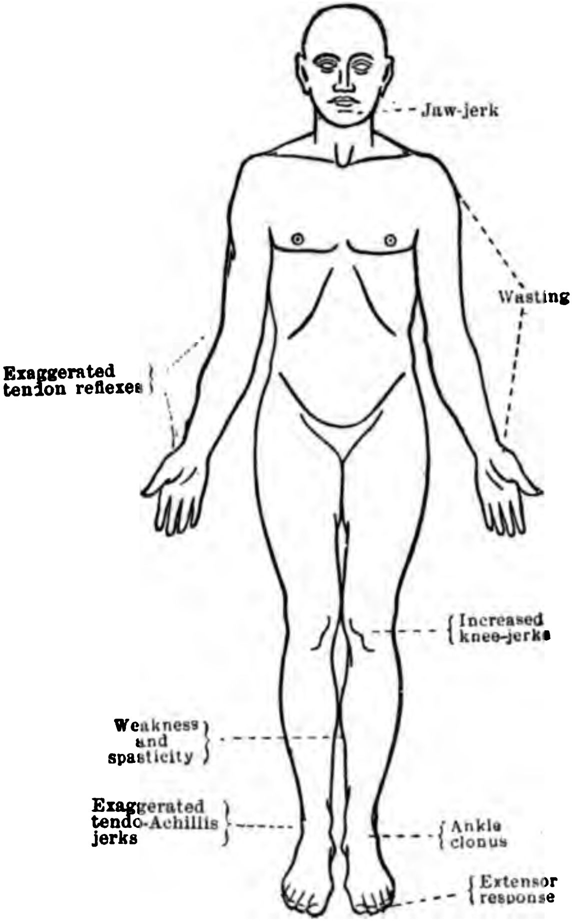

Fig. 62.—Scheme of the principal symptoms in amyotrophic lateral sclerosis.

Prognosis. — Amyotrophic lateral sclerosis runs much the same course as progressive muscular atrophy, and the dangers to life are similar in both diseases.

Treatment. — What has already been said of progressive muscular atrophy is applicable here, with the exception that strychnine may have to be withheld on account of its tendency to increase the rigidity.

ATAXIC PARAPLEGIA

Ataxic paraplegia is due to sclerosis of the lateral and posterior columns. As in primary lateral sclerosis, many of the cases which at one time appear to be examples of ataxic paraplegia develop other symptoms later, thus showing that the degeneration has not been confined to these two columns.

Etiology.—What has been said about the causation of primary spastic paraplegia is applicable here, but it should be added that toxins associated with extreme anæmias, diabetes, and other debilitating diseases seem to cause combined sclerosis rather than one which is limited to the lateral columns. Ergotism and pellagra may, among other symptoms, be accompanied by sclerosis of the posterior and lateral columns of the cord.

Symptoms.—The symptoms come on gradually, with weakness and spasticity of the lower limbs, just as in lateral sclerosis, and to these a variable degree of ataxy is added. Thus the clinical picture is one of lateral sclerosis with ataxy. In some cases there is a difficulty in controlling the sphincters; in some, too, sensation is blunted.

Prognosis.—The disease runs a chronic course, and, until the sclerosis extends beyond the lateral and posterior columns, it does not usually endanger life.

Pathology.—The spinal cord shows sclerosis of the lateral and posterior columns, and also, in many cases, of areas outside their boundaries. In the posterior columns the posterior external column in the neighbourhood of the posterior root zone escapes, in which it differs from the lesion of tabes. The reflex arc is thus not broken, and the sclerosis of the lateral columns accounts for the increased tendon reflexes.

Diagnosis.—The diagnosis must generally be provisional in view of the possible development of other symptoms. As in primary spastic paraplegia, a large number of cases prove later to be examples of disseminated sclerosis, as shown by the occurrence of such symptoms as nystagmus, ocular palsies, and pallor of the optic discs. In other cases the symptoms are the beginning of the subacute combined forms of degeneration, as described at p. 207.

Degenerative changes in the cord after a myelitis may cause symptoms that are practically indistinguishable from those of ataxic paraplegia.

Treatment.—No adequate method of arresting the degeneration is known. Treatment must be directed towards improving the general health and correcting any diseased condition from which the degenerative process seems to arise.

CHAPTER XXVI

FRIEDREICH'S DISEASE (HEREDITARY ATAXY*) AND HEREDITARY SPASTIC PARAPLEGIA

Etiology.—Although Friedreich's disease is known also as hereditary ataxy the symptoms are seldom transmitted directly from one generation to another. On the other hand, the disease is often found in several members of a family.

Both sexes are liable to suffer. The first symptoms may be noticed somewhere about the age of puberty, but they often begin at an earlier date. Occasionally an acute infective disease appears to have been the starting-point, but the degeneration can seldom, if ever, be definitely traced to any obvious cause, nor does it appear to depend on syphilitic taint.

Pathology.—The main lesions comprise degeneration and sclerosis of the posterior, lateral, and direct cerebellar tracts of the cord, and sometimes also of the antero-lateral tracts. The primary change is probably a degeneration of the nerve fibres, to which the overgrowth of connective tissue is secondary.

Symptoms.—When the parents are observant, as they are likely to be if one child after another is struck down, a change in the shape of the foot is often the first warning that anything is wrong. In others, clumsy movements of the legs or arms are the earliest signs. This clumsiness is due to the **ataxy** resulting from degeneration of the posterior and cerebellar tracts; it resembles that of tabes dorsalis, and is worse when the eyes are closed. The legs are generally first affected, and the child stumbles and falls about; later on, the disorder extends to the trunk and arms.

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* In some families, certain structures show a special disposition to atrophy prematurely. The reason for this is obscure, and can at present best be explained by assuming an inherent lack of vitality in the tissues, rather than the presence of an accidental toxæmia, for were the degeneration due to the latter it would not, probably, so frequently show itself in several members of a family. Premature degenerations of this kind occur in the nervous and muscular systems as well as in other organs of the body, and are included in the group to which the title of "abiotrophy" was applied by Sir William Gowers.

Weakness.—The pyramidal tracts are affected as well as the posterior columns; hence some weakness accompanies the ataxy, and the main symptoms in the legs are those of ataxic paraplegia.

Reflexes.—The continuity of the arcs for the tendon reflexes are usually broken at an early period of the disease, so that the knee-jerks are almost invariably lost.

The tendo-Achillis jerk and the deep reflexes of the upper limb likewise cannot be obtained.

When the plantar reflex is preserved it is often "extensor" owing to the degeneration of the crossed pyramidal tracts.

Deformities.—The shape of the feet alters, and assumes that known as "pes cavus," in which the arch of the foot is exaggerated and the toes become deformed as "hammer toes" owing to the contraction of the tendons, the extensor proprius hallucis often standing out very prominently (Fig. 63). A lateral curvature of the spine often develops.

The eyes.—There is usually a slow lateral *nystagmus* to be observed when the eyes are turned to one or other side. There is no Argyll-Robertson pupil, and optic atrophy has only rarely been observed.

Nodding movements of the head and a slurring, stuttering **speech** are both common.

Sensation.—In spite of the extent to which the posterior columns are sclerosed, there is seldom any appreciable loss of sensation, at any rate until the late stages of the disease. This shows how widespread are the paths through which sensory impulses are conducted up the cord.

Diagnosis.—A well-marked case can give rise to little difficulty, as the age at which it begins and the characteristic symptoms are very distinctive. The conditions most likely to cause confusion are juvenile tabes and disseminated sclerosis. Tabes acquired in later life can usually be excluded by the history.

The main points in which Friedreich's disease differs from *tabes* are set out in the following table:

	FRIEDREICH'S DISEASE	LOCOMOTOR ATAXY
Age of onset	About or before puberty	Later, except in rare cases of juvenile tabes.
Pains	Absent	Characteristic lightning pains.
Argyll-Robertson pupil	Absent	Frequent.
Optic atrophy	Absent (almost always)	Frequent.
Nystagmus	Present	Absent.
Loss of power	Present	Not common (unless quite local) till late stages of disease.

Disseminated sclerosis.—Disseminated sclerosis is in some cases differentiated with difficulty, since many of the symptoms are common to both affections; but the slow, unvarying progress of Friedreich's disease often contrasts with the variations met with in disseminated sclerosis, especially during the early stages. The coarser and more irregular tremor, the ocular palsies, and pallor of the optic disc all help to distinguish disseminated sclerosis.

Hereditary cerebellar ataxy.—Hereditary cerebellar ataxy of Marie very closely resembles Friedreich's disease; indeed, it is probably impossible to make a definite diagnosis in every case.

The main points which generally help to differentiate hereditary cerebellar ataxy are the later stage of onset and the preservation or increase of tendon reflexes, together with the absence of spinal curvature and of deformities of the feet.

Variations from type.—It is plain that with such a wide distribution of degeneration considerable deviation must be occasionally expected from the type originally described by Friedreich. Where the columns degenerate unequally it must sometimes happen that the symptoms due to an advanced condition of one predominate over those due to a slighter lesion of another tract, and this especially occurs when the lateral columns degenerate before the posterior and cerebellar. In such cases, spasticity and increased reflexes are the more prominent features. These patients generally complain first of deformity of the feet, and, from the youthful age and the history of the onset, the diagnosis of Friedreich's disease naturally suggests itself. Various other signs, e.g. a

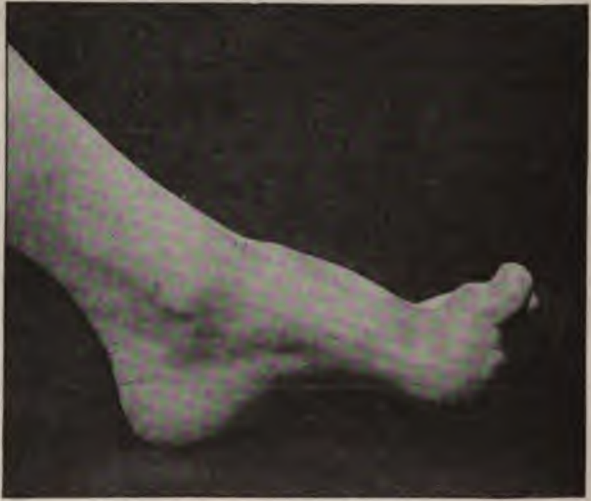

Fig. 63.—Pes cavus, from a case of Friedreich's disease.

certain degree of ataxia, nystagmus, and trophic ulcers of the feet, may lend confirmation to the diagnosis, though this last symptom is not usual in Friedreich's disease. The chief difficulty met with is the condition of the knee-jerks, which are exaggerated. This, according to most authorities, is absolutely against Friedreich's disease.

Thus we may be faced with a set of symptoms which closely resemble those of Friedreich's disease, but yet differ markedly from them in the character of the knee-jerks, the absence of which authors regard as so important a sign. Such cases may be considered as "atypical cases of Friedreich's disease," yet, considering the frequency with which they occur, it is more satisfactory to class them separately as cases of **hereditary spastic paraplegia.**

The term hereditary is somewhat unsatisfactory here as in hereditary ataxy, since this condition is likewise familial rather than inherited, but it is best to retain it since the term hereditary ataxy remains in use.

We have, then, in hereditary spastic paraplegia a disease which shows itself clinically by symptoms that appear chiefly to depend upon a premature degeneration of the lateral tracts, to which may be superadded symptoms due to some degeneration of the posterior columns and occasionally of other parts of the nervous system.

There is a close relationship between hereditary spastic paraplegia and Friedreich's disease, for the degeneration in both depends on some inherent lack of vitality; but the former can be distinguished from typical cases of Friedreich's disease by the predominance of the spastic symptoms, though occasionally, as might be expected, the two conditions practically merge into one another.

Prognosis.—The tendency of these spinal examples of abiotrophy is to progress slowly, but occasionally the cases seem to be stationary for years.

The author's impression is that the progress of disease in the hereditary spastic paraplegia type is slower and more likely to be arrested than in the Friedreich type.

Treatment.—No drugs are known to have any definite influence in checking the progress of the degeneration, but much can be done by maintaining the general nutrition with such drugs as cod-liver oil and iron, and by attending to the general health in every way. Under healthy conditions it is rational to assume that the progress of the disease is likely to be slower, and in some instances it may possibly be permanently arrested.

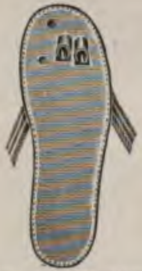
Fig. 64.—Device for dealing with hammer-toes.

Locally, the condition of the feet must be considered, and, as already mentioned, in cases of hereditary spastic paraplegia, where there is frequently, for a time, no ataxy, the deformity of the foot is the patient's chief complaint. Here it is that the hammer-toes are so troublesome, for they are distorted in such a way that they are always rubbing and pressing against the boot and are apt to become covered with very painful corns. While a suitable boot is very helpful in relieving this tendency, it is often necessary to devise some additional simple mechanical appliance. When the foot is flat on the ground, it will be observed that the toes are almost straight, and in that position would run very slight risk of being chafed by the boot; but the moment the foot is raised, up go the toes. The object of the mechanism illustrated in Fig. 64 is to keep the toes as far as possible in the position which they are still able to assume when the naked foot is placed flat upon the ground. A sole of firm leather is made to fit the under surface of the foot, and opposite the toes is a tape which passes through properly constructed holes in the leather sole, and thus enables the toes to be laced down. The tape, of course, can be changed as often as necessary. It is simple and inexpensive, and patients state that they find great benefit from it.

As a rule, the progressive nature of the disease, the number of tendons and the amount of fascia involved in the deformities, as well as the likelihood of difficulty in getting the parts to heal well, make these cases unfavourable for operation.

CHAPTER XXVII

SYRINGOMYELIA

OVERGROWTH of neuroglia and the formation of cavities in the spinal cord are the chief characteristics of syringomyelia.

Etiology.—The disease occurs in both sexes, and the symptoms are generally first noticed in the early years of adult life.

Injuries, inflammations, and vascular lesions occasionally seem to be the starting-point of the disease.

Pathology. — There are different causes for the formation of cavities in the cord. In some cases there seems to be a congenital developmental defect in the cord which results in distension of the central canal, the walls of which are lined with the usual epithelial cells. This condition is known as one of hydromyelia.

In other cases the proliferation of neuroglial tissue is the primary process, and the formation of cavities is then secondary to degeneration and bears no relationship to the central canal. It has further been suggested that the gliosis is due to some irritant poison, but this at present is a matter of speculation. The cavities, which vary both in shape and in position, thus accounting for the variations that are met with in the symptoms during life, are generally situated in the grey matter near the centre of the cord, but they often extend into the anterior horns, and sometimes into the posterior horns also. The walls of the cavity are generally formed by the proliferated neuroglial tissue (Plate 12).

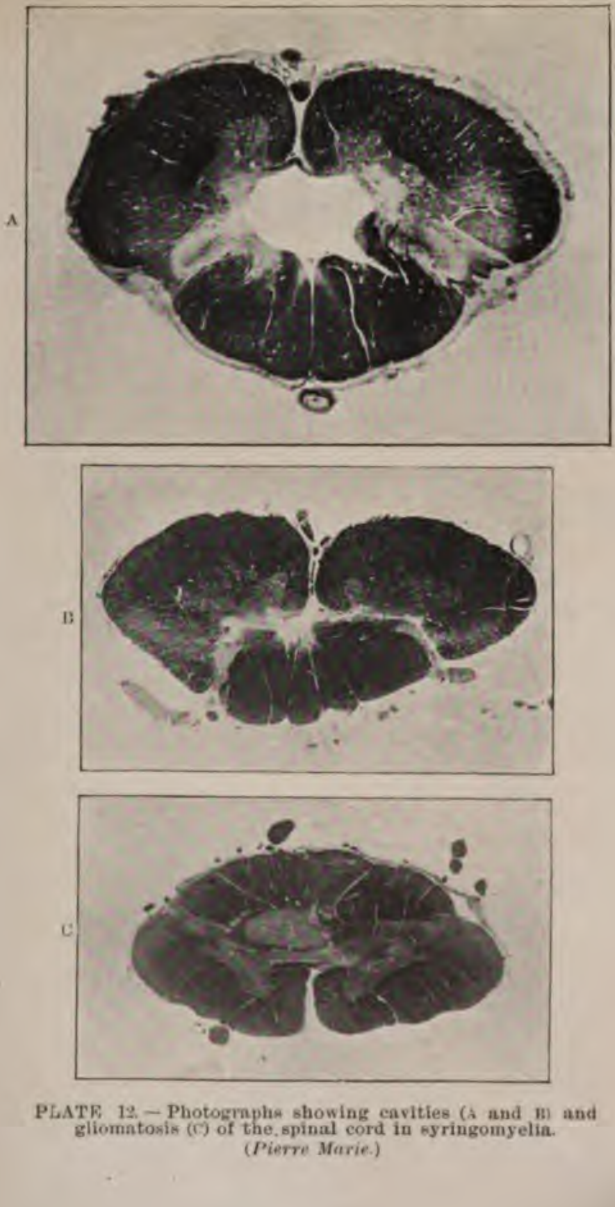

PLATE 12.—Photographs showing cavities (A and B) and gliomatosis (C) of the spinal cord in syringomyelia. (*Pierre Marie.*)

Symptoms. — The symptoms are due (1) to loss of cord substance, and (2) to pressure on surrounding parts by the proliferating neuroglia, which in some cases amounts to a definite tumour formation. The symptoms must, of course, differ with the position and size of the cavity and the degree of gliosis around it, but as the grey matter is especially liable to be affected there is a certain degree of uniformity in the main symptoms of any consecutive number of cases.

1. The lesions of the grey matter interrupt impulses of pain and temperature, hence there is usually *loss of sensation to heat, cold,* and *pain* (Figs. 65, 66), while the tactile sensibility frequently remains unimpaired.

2. Injury of the motor cells of the anterior horns occurs from extension of the cavity or from pressure of gliomatous tissue, and is followed by a slow, progressive *wasting of muscles.*

3. There are also *trophic lesions* of the skin, but the mechanism by which they are produced is uncertain.

The excavations are usually most extensive in the cervical region of the cord, so that the wasting of muscles is most prominent in the arms, while, at the same time, pressure on the lateral tracts often gives rise to some degree of weakness in the legs together with increase of knee-jerks, ankle-clonus, and extensor response of the plantar reflex.

The trophic disturbances are common (Fig. 67); some of them probably arise in the first place from burns and injuries which have not been felt. Spinal curvatures also occur.

Painless whitlows, arthropathies resembling those of tabes, vaso-motor disturbances of the skin causing localized anæmias or hyperæmias, excessive or deficient perspiration, bullous eruptions, local gangrene, and perforating ulcers, are all met with. The painless whitlows in conjunction with other nutritional disturbances of the hands were first described by Morvan, but it is generally considered that " Morvan's disease " is a variety of syringomyelia.

In some cases the morbid processes spread upwards to the medulla, and atrophy and weakness, sometimes one-sided, of the tongue, face, and palate may occur. Nystagmus is also occasionally seen.

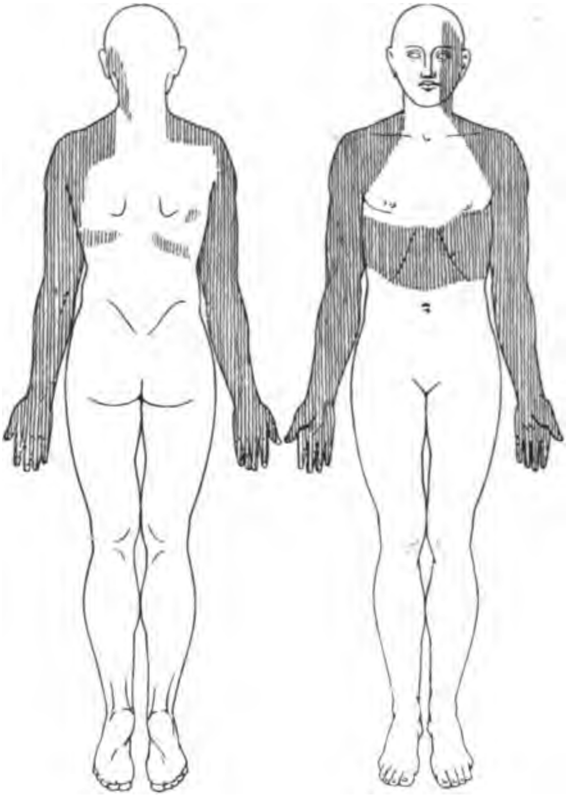

Fig. 65.—Diagram showing loss of sensation for heat and cold in a case of syringomyelia.

If the cavity is situated in the lower part of the cord the symptoms may be mainly confined to the legs. Occasionally a case may be met with in which there has been a sudden exacerbation of symptoms due to hæmorrhage having taken place into a cavity.

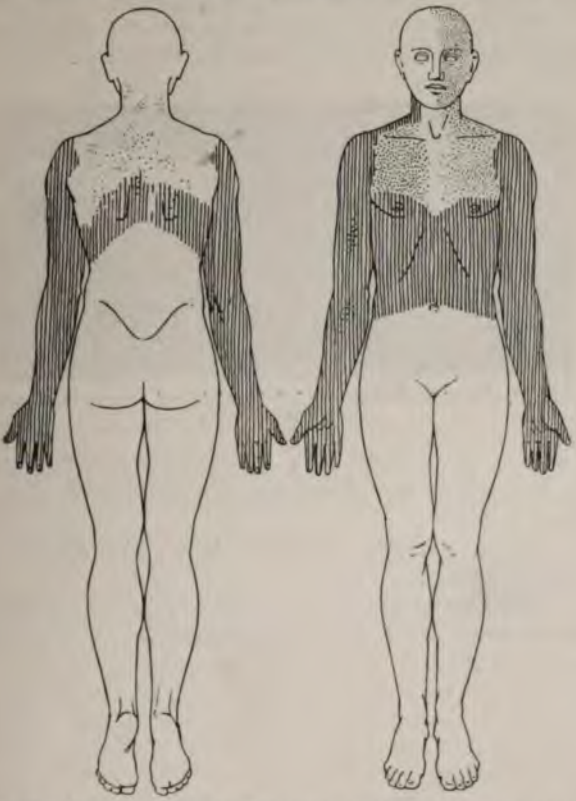

Fig. 66.—Diagram of the same case as in Fig. 65, showing areas over which sensations of pain were lost or blunted.

Diagnosis.—The wasting of the hands may lead to the diagnosis of ***progressive muscular atrophy***, unless the sensations for pain and temperature are tested, when the real nature of the condition will be apparent.

Where sensory disturbances are present alone, care must be taken not to put them down to ***hysteria***.

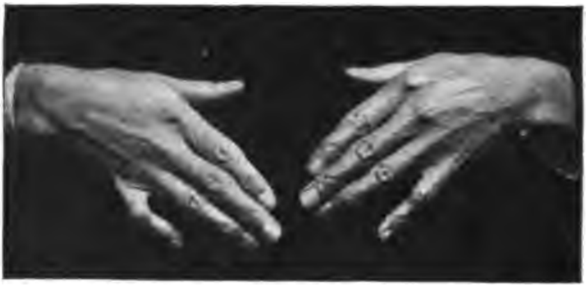

Fig. 67.—Trophic changes and deformities of fingers in a case of syringomyelia.

When the cavities are mainly in the posterior horns the symptoms may for a time simulate those of *locomotor ataxy*.

Prognosis.—The tendency is for the disease to progress slowly.

Treatment can only be directed towards keeping up the nutrition of the wasting muscles.

CHAPTER XXVIII

SPINA BIFIDA; HÆMATOMYELIA

SPINA BIFIDA

SPINA BIFIDA arises from maldevelopment of the laminæ of the vertebræ, with which malformations of the cord and its membranes are frequently associated.

Varieties.—The following six varieties may, according to Sir John Bland-Sutton,* be recognized:

(1) *Myelocele*, in which the central canal of the cord is open and from which the cerebro-spinal fluid continually drains away. This variety is more common in stillborn children, and those who are born alive usually do not survive more than a few days.

(2) *Syringomyelocele*, in which the central canal is dilated and forms the cavity of the protruded sac. This form is exceedingly rare.

(3) *Meningomyelocele*, in which the membranes and the cord both protrude, but the cord, instead of being dilated, is usually flattened on the posterior wall of the cyst. This is the commonest form.

(4) *Meningocele.*—In this variety there is a protrusion of the membranes only, the cord remaining normal.

(5) *Masked spina bifida* (spina bifida occulta), in which there is a deficiency of one or more of the vertebral arches which is not associated with any abnormality or protrusion of the cord or membranes.

(6) *Meningocele sacralis anterior*, a rare variety, observed only in girls and young women, in which the sac projects through a cleft in the bodies of the sacral vertebræ and presents in the pelvis.

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* "Tumours, Innocent and Malignant," 6th edition.

Spina bifida is apt to be associated with symptoms of hydrocephalus and syringomyelia. It also gives rise to various forms of talipes, and to nutritional disturbances such as perforating ulcer. Other deformities of developmental origin may also coexist.

Treatment.—In cases where it is possible to do so, excision of the sac is probably the best form of treatment. Many cases are best left untouched and protected by a suitable shield. In simple meningoceles good results are sometimes obtained by the injection of Morton's fluid, which causes the sac to contract. Morton's fluid consists of: Iodine gr. x, potassium iodide gr. xxx, glycerin ad ℥i. From ♏xv to ʒi is the amount usually injected at a time.

HÆMORRHAGE INTO THE SPINAL CORD (HÆMATOMYELIA)

Etiology.—Hæmorrhage into the substance of the cord may be associated with injuries to the spine, or it may occur into an area of the cord already diseased, e.g. in myelitis, tumours, and syringomyelia.

A primary spinal-cord hæmorrhage analogous to that met with in the brain is very uncommon.

Symptoms.—The symptoms depend upon the position and the extent of the hæmorrhage. Sudden pain in the back and paralysis are usually first met with, and in transverse lesions there is loss of sphincter control, together with anæsthesia of the parts below the lesion.

The extravasation of blood is often confined to the grey matter, and consequently the sensations for pain and temperature may be lost to a greater degree than the sensation for touch.

The cells of the anterior horns are also liable to be damaged, with resulting muscular atrophy.

If the patient recovers, there is a likelihood of some spastic paralysis below the level of the lesion, and of a permanent wasting of the muscles which are supplied from the anterior horns at the level of the lesion; thus wasting of the muscles of the arms is very liable to follow hæmorrhage in the region of the cervical enlargement. On the other hand, if the hæmorrhage is confined to the grey matter, there may be no paralysis below the level of the lesion.

Pathology.—There may be a single extravasation, or numerous hæmorrhagic foci may be present, and the condition of the surrounding cord largely depends on the existence or absence of previous disease. After injuries there is likely to be some hæmorrhage beneath the membranes as well.

Diagnosis.—The sudden onset of the symptoms suggests hæmorrhage. If the spine is injured the signs may be masked by the presence of extensive meningeal hæmorrhage. An important guide, when it can be demonstrated, is the loss of painful and thermic sensations, while the tactile sense is retained, thus pointing to a lesion of the grey matter. The existence of previous disease in the cord must be decided by the history.

Prognosis.—The prognosis depends on the cause and the extent of the hæmorrhage. In cases of injury where the hæmorrhage has been slight, patients often do well, although they may have permanent paralysis if cells with axons innervating important muscles be damaged.

Treatment.—The patient should be kept as quiet as possible; an ice-bag should be applied to the spine. The bowels should be opened. If necessary, morphia may be given to relieve pain. Ergot has been recommended on account of its supposed power of checking the hæmorrhage.

CHAPTER XXIX

TUMOURS OF THE SPINAL CORD

Tumours of the spinal cord may be divided into two classes, according as they arise within or without the substance of the cord.

The chief tumours that arise *within* the substance of the cord (intramedullary tumours) are gummata, sarcomata, gliomata, and tuberculous growths. Probably the commonest of these intramedullary tumours are gummata.

Gliomata do not occur so often in the cord as in the brain, and intramedullary tuberculomata are decidedly uncommon.

Outside the cord (extramedullary tumours), sarcomata, carcinomata, psammomata, lipomata, fibromata, gummata, and hydatids are found; and occasionally osteomata have been observed to press upon the cord. Carcinomata occur chiefly in the vertebræ, and are in most cases secondary to disease elsewhere.

Sarcomata generally arise from the pia mater, and are sometimes widely diffused over the membranes of the brain as well as of the cord. (Plate 13.) Their place of origin is then often difficult to determine, the disease at times appearing to have spread upwards from cord to brain, and at other times from brain to cord. In the latter case the extension may be due to infection of the cerebro-spinal fluid by a primary growth situated high up in the nervous system and exposed to the stream of cerebro-spinal fluid. (Stanley Barnes.)

Psammomata (dural endotheliomata) also grow from the pia mater. (Fig. 68.)

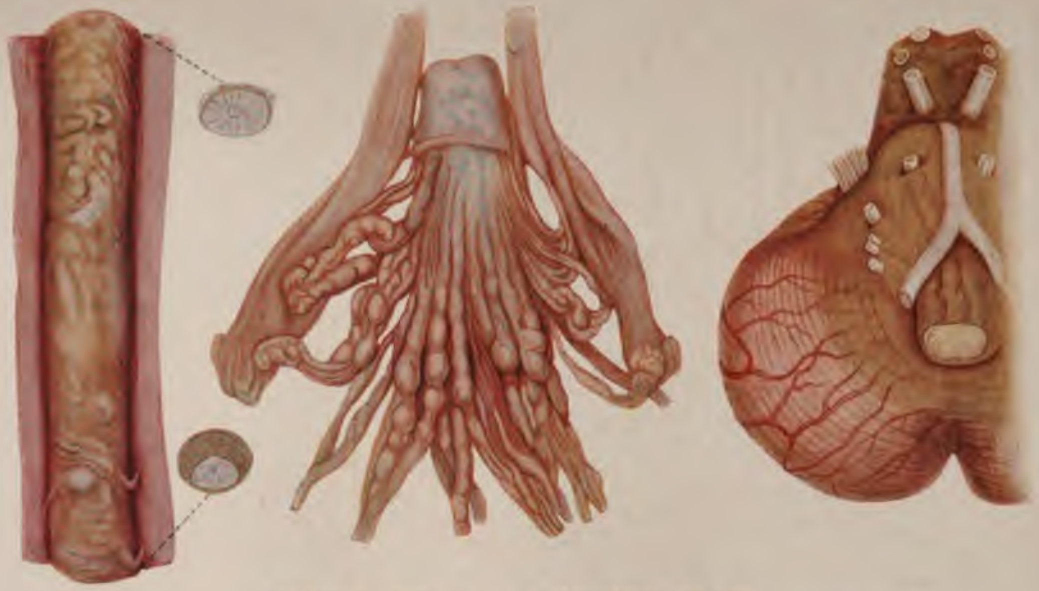

PLATE 13.—Diffuse sarcoma of the brain and cord.

(*From a case reported in the Transactions of the Pathological Society by Drs. Coupland and Pasteur.*)

Lipomata usually grow from the areolar tissue outside the dura mater; they may also arise on its inner surface.

Fibromata sometimes arise in connection with nerve roots; they are referred to in Chapter XV. (p. 162).

Gummata of the membranes are often ill-defined tumours, and are frequently accompanied by extensive meningeal infiltration and endarteritis.

Hydatids are important causes of compression of the cord. They nearly all grow in the areolar tissue between the dura mater and the bone. Of 44 cases tabulated by Schlesinger (quoted by Bland-Sutton), 39 were extradural and only 5 intradural.

Symptoms.—The symptoms differ somewhat according as the tumour is situated outside or inside the substance of the cord.

When outside the cord (*extramedullary*) a tumour produces symptoms by *pressure* (1) on the nerve roots and (2) on the cord itself.

The nerve roots generally suffer first, so that the patient complains of *pain*, which tends to follow the root distribution, and which for a time is generally unilateral. As the tumour grows, it presses on the cord, and also perhaps on the anterior nerve roots, and thus to the pain are added *weakness* and *loss of sensation*, which in the early stages may be of the Brown-Séquard distribution.

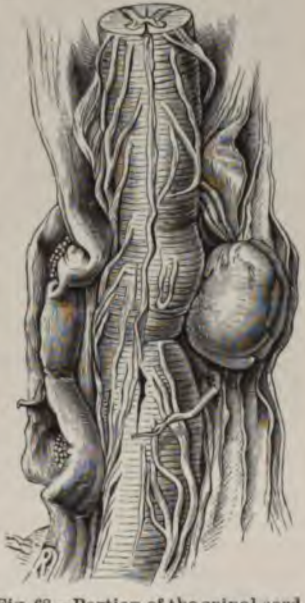

Fig. 68.—Portion of the spinal cord with a psammoma situated at the level of the intervertebral disc between the 10th and 11th thoracic vertebræ. From a woman 46 years of age. (*Sir J. Bland-Sutton; Museum, Middlesex Hospital.*)

The weakness below the level of the tumour will be of upper neurone type (spasticity, increased knee-jerks, extensor response of the plantar reflex, with no wasting or changes in the electrical reactions of muscles), while, at the level of the lesion, compression of one or more anterior roots may cause some paralysis and wasting over the area of their distribution.

The main alterations in sensation are anæsthesia over the areas supplied by the compressed posterior roots, to which may be added subsequently a more generalized anæsthesia below the lesion, due to pressure of the growth on the sensory columns of the cord. As a rule, the loss is more or less equal for all kinds of sensation. At the level of the tumour there may be a zone of hyperæsthesia. With increasing compression a complete paraplegia develops with loss of power over the sphincters.

When the tumour grows in the substance of the cord (*intramedullary*) there is usually less pain, while paralysis and anæsthesia are more likely to be early symptoms.

The distribution of the weakness and of the alteration in sensation during the early stages of an intramedullary growth depends upon the locality of the cord in which the new growth originated. If, for instance, the substance of the grey commissure is destroyed, the signs will at first very probably simulate those of syringomyelia, viz. loss of thermal and painful sensibility with preservation of tactile sensation. To these signs may be added some muscular wasting, as the pressure is felt by the cells in the anterior horns.

On the other hand, if the tumour grows for a time in one half of the cord, the signs will tend towards those of the Brown-Séquard form of paralysis, viz. loss of power below and on the same side as the tumour, with loss of sensation chiefly on the opposite side (*see* p. 21).

As the growth invades more and more of the cord, these early distinctions become masked by the onset of more generalized paraplegia associated with a variable amount of anæsthesia below the lesion.

The symptoms are then usually those of a transverse lesion of the cord, viz. spastic paralysis, anæsthesia, and loss of power over the sphincters below the lesion.

Tumours of the cauda equina.—The symptoms of tumours of the cauda equina, since the paresis is of the lower neurone type, are mainly those of flaccid paralysis with some wasting and electrical changes. The effect on the different structures varies with the level of the lesion. Thus, the knee-jerks may be retained if the fibres going to the anterior crural nerve are unaffected; the degree of sphincter control, too, varies in different cases.

Compression of the cord from tumours, especially at the lumbar region and cauda equina, may cause changes in the cerebro-spinal fluid known as Froin's syndrome, the chief characteristics of which are yellowish discoloration, heavy coagulation, and an increase in the number of lymphocytes. Discoloration of the fluid only is not pathognomonic of compression, as it may arise as the result of an old hæmorrhage.

Diagnosis.—The recognition of tumours at an early stage is very difficult, for the cause of the pain is often indefinite, and, in the absence of other signs, is liable to be attributed to such conditions as *muscular rheumatism*, *neuritis*, and *sciatica*. Suspicion should, however, be aroused by the constancy of the pain, its tendency to follow an area of root distribution, and especially by the "girdle" character which it often assumes. This suspicion will be confirmed by the discovery of progressive weakness and sensory disturbance. When slow-growing cases of intramedullary growth are not accompanied by pain they may be difficult to distinguish from degenerative processes in the cord.

The diagnosis of a spinal tumour having been made, it is necessary to try to decide whether it is situated within or without the cord.

In some cases the early implication of the roots, followed subsequently by signs of gradual compression of the cord, are almost certain indications that the growth is extramedullary; in others, the dissociated anæsthesia, together with the absence of signs of pressure

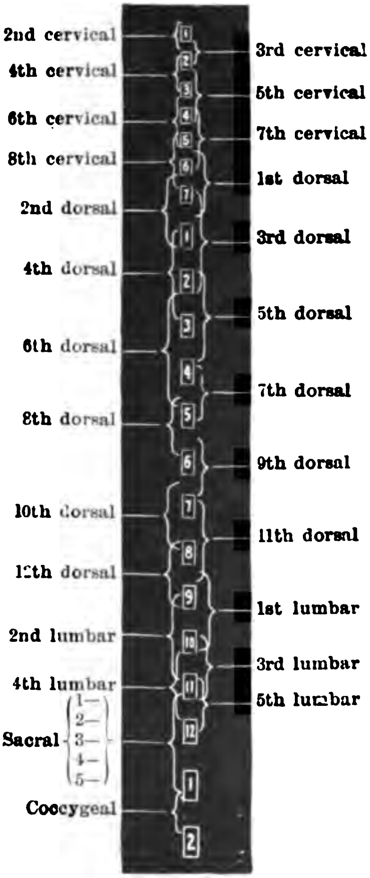

Fig. 69.—Diagram showing relations of the root origins of the spinal nerves to the spines of the vertebræ. (*After R. W. Reid, "Quain's Anatomy."*)

on the roots, makes it almost equally certain that the tumour is growing in the substance of the cord. But between these two groups there are many cases in which diagnosis is difficult, especially if the patient has already passed the early stages of the disease.

The next point to decide is the level at which the tumour is situated. The guide to this is to be found in the upper limits of the signs. When the anæsthesia and pain are due to pressure on roots their areas must be very carefully mapped out and identified with their particular roots, and the uppermost nerve root that is affected should demarcate the upper level of the tumour. Any local loss of power due to pressure on anterior roots can be utilized in a similar manner for localizing purposes.

When the anæsthesia is of intramedullary origin, its upper level will likewise denote the position of the tumour, but any zone of hyperæsthesia that may exist about the level of the lesion must be taken into consideration, since its presence will indicate root irritation, and will show the necessity of locating the growth rather higher than the anæsthesia indicates.

When the sensory signs are taken to indicate the level of the lesion, it must be remembered (1) that the origin of the roots from the cord is not level with their foramina of exit, and (2) that the spines of the vertebræ, owing to their obliquity, are not on a level with their corresponding bodies. Unless these points are kept in mind there will be a likelihood of the localization being made too low. The relations of the nerve roots to the vertebral spines are shown in Fig. 69.

Treatment.—If the tumour is syphilitic, or if there is a reasonable doubt regarding its nature, an efficient course of antisyphilitic treatment should be tried. If the symptoms do not abate, the question of the removal of the neoplasm should be considered without delay. The chances of success in an operation for this purpose are naturally greatest in the case of extramedullary growths. It is, however, also found possible

is described in detail by
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CHAPTER XXX

SPINAL MENINGITIS AND MENINGEAL HÆMORRHAGE

SPINAL MENINGITIS

FOR purposes of classification, the cases of spinal meningitis may be divided into inflammations of the dura mater (pachymeningitis) and of the pia mater (leptomeningitis), although it necessarily often happens that no distinction can be drawn between them either at the bedside or in the post-mortem room.

Pachymeningitis.—Inflammation of the dura mater may commence on the inner or outer surface, and the commonest causes are syphilis, injury, and extension of inflammatory processes from neighbouring parts, e.g. from tuberculous disease of the vertebræ.

One form (*pachymeningitis hypertrophica*), in which the dura mater is greatly thickened, is especially apt to occur in the cervical region of the cord, and to cause wasting and pains in the arms.

Leptomeningitis.—In leptomeningitis the inflammation is at first seated in the pia mater and arachnoid.

Acute infections, as from tubercle, pneumonia, septicæmia, and epidemic cerebro-spinal meningitis, are the chief causes of this variety.

A local *serous meningitis* in which a collection of fluid in the subarachnoid often presses on the cord and resembles a tumour may also occur.

Symptoms.—The symptoms of spinal meningitis arise mainly from compression and irritation of the anterior and posterior nerve roots and also from extension of the inflammation to the structures of the cord. Hence the symptoms will differ with the position and intensity of the process. In *hypertrophic meningitis* of the cervical region, to which allusion has already been made, there is generally a slow strangulation of the nerve roots of the arms with consequent pains and wasting; and perhaps at a later date there may be signs of pressure on the cord giving rise to the effects of an incomplete transverse lesion.

In *acute leptomeningitis* the signs of a general infection, such as fever, rapid pulse, and perhaps rigors, are also usually present. The symptoms of irritation of the nerve roots are very prominent, and are shown by pain over the spine which radiates down the limbs, together with spasms, cramps, and rigidity of the muscles. The head is often drawn back, the back arched, and the abdomen retracted.

The control over the sphincters is frequently impaired, and there may be other signs that the inflammation is extending into the substance of the cord. Bedsores and crops of herpes are likely to occur.

In the more *chronic* forms, pain is generally a prominent feature and the motor symptoms are less severe.

In many of the infective cases cerebral meningitis is also present, and to a considerable degree masks the spinal symptoms.

The principal characters of syphilitic meningitis are described in Chapter XXXI.

Diagnosis.—In its **acute forms,** spinal meningitis has to be distinguished from *myelitis.*

Reliance must be placed chiefly on the signs of irritation of the nerve roots which are so typical of meningitis, while paralysis is likely to be more marked in a lesion of the cord. Of course the two conditions may be present at the same time.

In severe cases the symptoms may be suggestive of *tetanus*, but they are not in keeping with the onset of tetanus, which usually begins in the muscles of the jaw and neck.

The symptoms of *hæmorrhage into the meninges* are more rapid in their onset.

In the **chronic cases** the wasting of muscles may resemble that of *progressive muscular atrophy*, but the presence of the pains and anæsthesia generally shows that the disease is not confined to the motor system. In other instances, especially in the localized serous variety, there is difficulty in distinguishing meningitis from *tumour*, which the pressure symptoms often closely resemble.

Examination of the fluid obtained by lumbar puncture may be an important aid to diagnosis, especially in the acute cases.

Prognosis.—The prognosis of acute spinal meningitis is not good, for the dangers of extension to the region of the medulla and brain and to the substance of the cord are all great. In a certain number of cases the symptoms subside, leaving behind them a variable amount of permanent damage, and in a few instances a good recovery takes place. In the chronic cases the outlook varies with the position and cause of the inflammation. Where the origin of the disease is outside the dura mater, as it is in spinal caries, the results are often satisfactory if the original focus of disease be removed. Some of the syphilitic cases, of course, do very well, but, as mentioned elsewhere (p. 274), permanent cure even in these cannot be relied upon.

Persistence or return of symptoms during treatment and extension of the disease are bad signs.

Treatment.—Whenever syphilis is suspected, salvarsan or mercury should be administered. Potassium iodide may be given in full doses at the same time. Iodide is also undoubtedly sometimes useful in cases which apparently are not of syphilitic origin.

When the disease is chronic and has begun outside the dura mater, the pressure on the cord may sometimes be relieved by performing laminectomy.

In the acute infective cases an ice-bag may be applied to the spine, though in many cases warm applications are more comforting. Counter-irritation is useful, but care must be taken not to set up bedsores, especially if myelitis is present at the same time.

Mercurial inunction may be used, as in myelitis, for, even though there is no history of syphilis, mercury seems often to exercise a beneficial influence over the inflammatory processes. Morphia must be given if required.

The same care in nursing and attention to the bladder are necessary as in myelitis.

MENINGEAL HÆMORRHAGE

Etiology.—Hæmorrhage into the membranes of the cord may be associated with hæmorrhage into the brain, and is then usually of secondary consequence. Injury of the spine is sometimes accompanied by some hæmorrhage, which is more often outside the dura mater. Acute fevers, degenerated vessels, and severe anæmias are occasionally the causes of hæmorrhages into the membranes of the cord as elsewhere.

Symptoms.—The symptoms are those of irritation of nerve roots and pressure on the cord; hence they are much the same as those of an acute spinal meningitis, except that they arise more suddenly.

There is severe pain in the back and along the course of the nerve roots, accompanied by a variable degree of spasm of muscles, and, when the cord is compressed, there is a more or less complete paralysis below the lesion. The deep reflexes are at first diminished, but later become exaggerated, and efficient control over the sphincters is often lost.

Prognosis.—The prognosis depends in the first place on the amount of blood extravasated and on the effects which it produces on the cord. The extent of the inflammatory and other after-effects remains to be seen, so that in every case the prognosis must necessarily be one of caution.

Diagnosis.—Hæmorrhage into the membranes of the cord is distinguished from meningitis by the suddenness with which the symptoms originate and by the absence of the more general symptoms that usually accompany acute inflammations of the cord. Otherwise they closely resemble each other.

The presence of an injury or some other distinct cause makes the diagnosis easier. It may be difficult sometimes to distinguish meningeal hæmorrhage from hæmorrhage into the substance of the cord, especially as both may be present at the same time. In the latter there is a tendency for the blood to be extravasated into the grey substance, and so to destroy sensibility to pain and temperature, leaving that to touch unimpaired. The diagnosis may also be assisted by the appearance of the fluid, as shown by lumbar puncture.

Treatment.—If there are reasons to believe that the blood is compressing the cord, the tension should be relieved by performing laminectomy. In other cases absolute rest should be enjoined, and an ice-bag should be applied to the spine. Calcium chloride may be given to promote clotting.

SECTION V.—SYPHILIS OF THE NERVOUS SYSTEM

CHAPTER XXXI

INTERSTITIAL AND PARENCHYMATOUS SYPHILIS

SYPHILITIC disease of the nervous system may be divided into two groups according as the spirochæte attacks chiefly (*a*) the connective tissues, vessels, and meninges, or (*b*) the nerve substance itself.

The lesions of the first group are known as ***interstitial*** syphilis, while those of the second are designated *parenchymatous* and in most instances correspond to the lesions formerly described under the title of parasyphilis.

The term parasyphilis was first used by Fournier, to indicate those late results of syphilis which were thought not to be direct manifestations of the disease in the sense that secondary and tertiary lesions were considered to be, but indirect results dependent in some way on the loss of vitality resulting from the presence of toxins.

The two parenchymatous forms to which most attention has been given are tabes and general paralysis of the insane, though, as will be mentioned later, modern investigation has shown that Fournier was correct in including several other nervous diseases in this category. Evidence of the syphilitic nature of tabes and general paralysis has gradually accumulated, and in later years has been strengthened in this country by the pathological work of Mott and his insistence on the fact that the two diseases are identical in their pathology. The discovery of the Wassermann test forged a further link in the chain of evidence, and any remaining doubt was finally dispelled by Noguchi's discovery of the spirochæte in the tissues of the brain and cord.

Thus the diseases formerly classed as parasyphilis, and thought to be indirectly due to syphilis, are now known to be directly due to the activity of the specific organism, the *Spirochæta pallida* (*Spironema pallidum*) and are to be regarded as manifestations of the activity of this organism. In these circumstances the term parasyphilis is no longer required, and while it is not possible to make any sharp division between lesions of the connective tissue and those of the nerve elements, owing to extension of disease from one to the other, it has nevertheless for general clinical purposes become customary to divide syphilis of the nervous system into the two categories, viz. parenchymatous and interstitial, according to whether the nerve elements or the supporting connective tissues are predominantly the seat of the disease.

The diagnosis of both interstitial and parenchymatous syphilitic affections of the nervous system is greatly assisted by microscopical examination of the cerebro-spinal fluid and by the Wassermann reaction.

The cerebro-spinal fluid.—In the cerebro-spinal fluid of a normal person there are scarcely any lymphocytes to be seen. In syphilis of the meninges, brain, and cord, lymphocytes are frequently found in large numbers, which vary from time to time according to the activity of the disease, so that from the variation of their numbers it is sometimes possible to gain some idea of the effects of treatment; but, as lymphocytosis occurs in other conditions besides syphilis, it must not by itself be taken to be diagnostic of this disease.

Changes in the chemical qualities of the cerebro-spinal fluid, e.g. an increase of proteid substance, have been described as a result of syphilis; but this cannot be regarded as pathognomonic of syphilis, for it has been found also during certain stages of dementia præcox.

The Wassermann test.—A positive reaction of the serum or of the cerebro-spinal fluid to the Wassermann test indicates that certain chemical changes in the one or the other have been produced by the action of the spirochætes on the tissues of the body, and it is now generally accepted that, where the tropical and other diseases in which the reaction is positive can be ruled out, a positive reaction in either of these fluids is a definite indication of syphilitic infection.

In locomotor ataxy the reaction both in the serum and in the cerebro-spinal fluid obtains with considerably less frequency than in general paralysis, owing probably to the more chronic character of the disease and its greater liability to periods of quiescence.

While the Wassermann reaction can usually be modified rapidly by treatment in interstitial syphilis of the nervous system, it is only with great difficulty or not at all that it can be altered in the parenchymatous forms, on account apparently of the resistance which the nerve tissues offer to the penetration of drugs that affect the spirochætes.

McIntosh, Fildes, Head and Fearnsides * have shown that the importance of the Wassermann spinal-fluid reaction does not necessarily end in simply establishing the diagnosis of syphilis, for the behaviour of the reaction can be further used to distinguish certain types of syphilis and arrive at their prognosis. For example, in meningovascular disease of the brain and spinal cord, these authorities consider that the key to the reaction of the cerebro-spinal fluid lies in the presence or absence of inflammatory changes in the meninges of the spinal cord and brain stem, and that disease of the brain alone above this level will not give the reaction.

This point, if established, forms, as the authors insist, an important possible distinguishing feature between cerebral meningo-vascular syphilis and general paralysis, for while the cerebro-spinal fluid of general paralysis practically always gives a positive reaction, that of cerebral meningo-vascular syphilis will be negative unless complicated by some lesion in the brain stem and cord. Moreover, when the reaction is positive its significance can be further gauged by the effects of treatment, for while the reaction of meningo-vascular syphilis and its clinical signs can usually be modified by a course of salvarsan, those of general paralysis rarely undergo any great change.

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* *Brain*, xxxvi. 1913 and xxxvii. 1914.

INTERSTITIAL SYPHILIS

While lesions of this form of cerebro-spinal syphilis may appear at any stage, it is important to remember that they may occur even within a very few months of the acquisition of a primary sore, and that they are very commonly seen within a period of two years from the date of infection. According to Naunyn, 48 per cent. of the cases occur within the first three years. That the nervous system is often affected in the very early periods of the disease, although there may be no clinical signs, is shown by the possibility of finding pleocytosis, or even, rarely, a positive Wassermann reaction, in the cerebro-spinal fluid during the primary stages of the infection. Men are attacked more frequently than women, and the cerebral structures more often than the spinal, and when the disease is apparently localized in the cord there are generally some signs to suggest that the brain has not altogether escaped.

INTERSTITIAL SYPHILIS OF THE BRAIN

The structures chiefly affected are the vessels, the membranes, and the neuroglia. It is usual to find the membranes and vessels affected at the same time, forming the condition termed *syphilis meningo-vascularis* (Head and Fearnsides). Endarteritis is often followed by thrombosis and subsequent softening of brain tissue. A diffuse gummatous meningitis, through which the brain is damaged by pressure or extension of the inflammation, is the commonest affection of the membranes. Less commonly, definite discrete gummata are found.

Syphilis meningo-vascularis very frequently begins at the base of the brain; the cranial nerves are soon damaged, and a special feature of the disease is the random way in which they are picked out. The nerves supplying the ocular muscles are particularly liable to suffer, and ocular palsies, often of transient character, are among the most common symptoms of basal syphilitic meningitis. Pupillary changes, apart from tabes and general paralysis—e.g. inequality in size and shape and deficiency in reaction to light—are common. Optic neuritis is frequently present, and when the disease is situated in the middle fossa there may be other disturbances of vision due to pressure on the optic chiasma. In disease of the posterior fossa the spinal accessory and hypoglossal nerves are likely to be affected.

A gumma may, of course, give rise to the general and local signs common to other forms of cerebral tumour.

Variation of the symptoms, intense headache which is usually worse at night, vomiting and early implication of the cranial nerves, are among the signs that suggest syphilitic meningitis. Among other symptoms of which the patient may early complain, Head and Fearnsides mention shivering attacks, with or without fever, neuralgic pains of radicular origin, and general changes of character, e.g. want of concentration and loss of memory.

Where the function of the frontal lobes is modified, the symptoms may closely simulate those of general paralysis of the insane, and the resemblance may be enhanced by the addition of convulsions, more especially when the infiltration of the membranes extends over the motor areas. Such cases occurring in the secondary period are probably due to a spread of the disease to the parenchymatous structures, and their differentiation from the later parenchymatous forms is an important matter. Irregular patches of infiltration, accompanied by symptoms which frequently disappear, may suggest the presence of disseminated sclerosis, the more so if at the same time patches of a similar kind are disturbing the functions of the spinal cord. The clinical picture of cerebral syphilis may show considerable changes from time to time, owing to the variation in vascularity and intensity of inflammatory processes in the diseased parts. Its resemblance to other disorders may thus be greater or less according to the stage which the disease has reached at any particular time; and in the early stages, when there are no definite signs, care must be taken to differentiate it from simple neurasthenia, many of the manifestations of which it may closely simulate.

INTERSTITIAL SYPHILIS OF THE SPINAL CORD

As in the case of the brain, the structures chiefly affected are the neuroglia, the meninges, and the blood-vessels. (Fig. 70.)

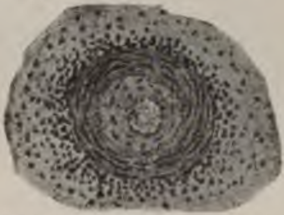

Fig. 70.—Endarteritis and periarteritis of a meningeal artery from a case of syphilitic meningomyelitis. (*R. T. Williamson.*)

The commonest lesion is that known as **meningomyelitis,** in which there is a diffuse inflammation of the neuroglia, vessels, and membranes.

The meninges become diffusely infiltrated with a gummatous substance over an area of varying extent, and at the same time the walls of many of the arteries which pass into the cord are thickened by an inflammatory process of a similar nature, which narrows or altogether blocks their lumen. The meningeal thickening causes symptoms by compression of the nerve roots and other structures around, while the narrowing of the blood-vessels impairs the circulation in the cord. The posterior arteries of the cord (Figs. 71, 72) are generally most affected, and so the effects of circulatory disturbance are chiefly visited upon the posterior columns and the pyramidal tracts, with the result that the main symptoms are those of paraplegia, together with some degree of loss of sensation, to which diminished control over the bladder is often added.

Thus, clinically, this condition shows itself in the form of an incomplete transverse myelitis in which the symptoms, while similar to those of other forms of myelitis, are apt to vary in their intensity from time to time as the condition of the vessels allows more or less blood to reach the nerve structures.

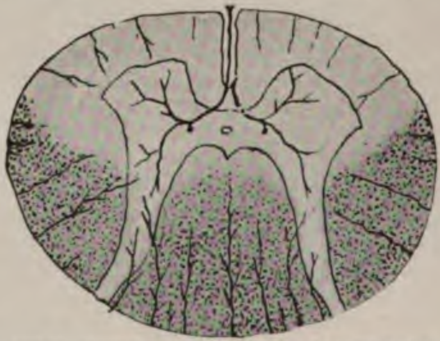

Fig. 71.—Section showing distribution of the anterior and posterior arterial systems of the cord. The part shaded by dots shows the area supplied by posterior arteries of the cord. (*R. T. Williamson.*)

Erb has described a group of cases, of gradual onset, in which the motor columns suffer out of proportion to the sensory tracts, the control of the sphincters being also frequently weakened. It seems probable that in some cases of this description the lesion is due to a parenchymatous systemic degeneration of the lateral and posterior columns and not to a meningomyelitis, but the distinction from a clinical point of view, apart from the results of treatment, may be very difficult, and indeed it is possible that systemic degeneration and a diffuse myelitic process may coexist.

Acute syphilitic myelitis.—Sometimes syphilitic changes in the cord come on very rapidly, and give rise to the clinical picture of acute transverse myelitis; indeed, many cases included under the latter title are no doubt instances of rapid softening of the cord consequent upon syphilitic endarteritis and thrombosis. The outlook in such cases is not good.

Gummata.—Isolated gummata are not common either in the membranes or in the cord itself. Their symptoms are similar to those of other tumours, and when situated in the substance of the cord they are apt to produce the Brown-Séquard form of paralysis, or symptoms which are closely allied to it.

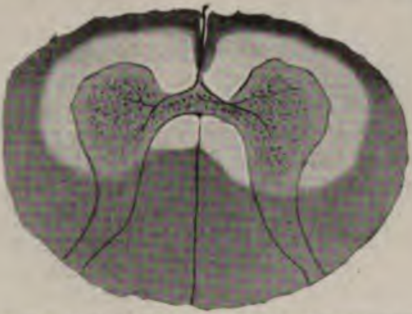

Fig. 72.—Diagram showing division of a transverse section of the cord into three zones according to the arterial supply: (i.) Central zone (dotted) in the centre of the grey matter which is supplied by the anterior median or central arteries of the cord; (ii.) a peripheral zone (shaded) supplied by the peripheral arteries of the cord; (iii.) a third zone (pale) supplied by both central and peripheral arteries. (*R. T. Williamson.*)

Diagnosis.—The combination of manifestations that are due to disease of the membranes and of the cord, together with the variability of the symptoms, the history of a primary infection when it can be obtained, and the results of the Wassermann tests of the blood and spinal fluid, are some of the main points to which attention should be paid. A careful examination of the back should be made in order to exclude the presence of caries, and the possibility of the existence of a tumour other than a gumma must be remembered.

Irritation and compression of the posterior roots by the thickened meninges may cause symptoms resembling those of *tabes*, but the rapidity with which the symptoms arise, together with the additional signs of meningitis and extension of the disease to the more central structures of the cord, generally makes the diagnosis clear.

Prognosis.—When the circulation in any part of the cord becomes almost arrested there is great danger of softening, followed by a sclerosis, which permanently damages the cord.

The patient is also very liable to relapses.

The course of the disease cannot be predicted until the effects of antisyphilitic treatment are seen, but it may be said generally that, when the symptoms are of recent origin, the results are fairly favourable. In some instances the symptoms disappear with great rapidity; in others there is permanent disablement from the effects of secondary degenerations; in others again the paraplegia becomes complete and complicated by bedsores and cystitis, with subsequent death from exhaustion.

PARENCHYMATOUS SYPHILIS

Parenchymatous syphilis, as we have seen, is the term denoting the results of infection of the nervous elements of the brain and cord by the spirochætes. The condition is mainly characterized by degeneration of tracts of neurones, and clinically therefore the symptoms differ in different cases according to the grouping and the order in which the degeneration takes place.

The combination of symptoms most widely recognized is that of tabes, general paralysis, and the mixed cases of tabo-paresis, but there is no reason why other combinations should not take place, and Head, Fearnsides,* and others have pointed out that some clinical forms of progressive muscular atrophy, lateral and combined sclerosis, primary optic atrophy and periodic epileptiform attacks may equally well be manifestations of the disease.

An important fact, concerning which probably all observers are agreed, is that in parenchymatous syphilitic affections of the nervous system the symptoms of the original infection have often been slight, and even so insignificant as to escape the attention of the patient. The difficulty which frequently occurs in obtaining any history of infection is well known, and Mott,† in 600 post-mortem examinations of cases of general paralysis, observed that external marks of syphilis in the shape of lesions of the skin, bones, and viscera were relatively few in number. The presence of gummata or scars of gummata in the viscera is also rare, and in a considerable number of cases the only internal evidence that can be found of previous syphilis is nodular fibrosis of the aorta. It has been suggested that these cases are associated with a type of spirochæte which shows a peculiar tendency to attack the nervous system to the exclusion of the other tissues of the body, a view which recent experimental work seems to support.

The difficulty which often exists in obtaining evidence of syphilitic infection in tabes and general paralysis is now largely counteracted by means of the Wassermann tests of the blood and spinal fluid, which give a positive result in almost every instance of general paralysis and in a large percentage of the cases of tabes.

The first stage of the syphilitic infection having passed, there generally ensues a quiescent period of considerable length (an average of about twelve years in the case of tabes and general paralysis), in which the spirochætes remain latent, and it has been suggested that they may during this period possibly assume a granular form.

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* "The Clinical Aspects of Syphilis of the Nervous System in the Light of the Wassermann Reaction and Treatment with Neosalvarsan," by Henry Head and E. G. Fearnsides; *Brain*, 1914, vol. xxxvi., Pt. i.

† *Archives of Neurology and Psychiatry*, vol. vi.

When activity recommences, degenerations begin to make their appearance, and the manifestations of parenchymatous syphilis arise.

As to the nature of the reactions which precede the degenerations, somewhat different views are taken. That most commonly accepted, due largely to the work of McIntosh and Fildes,[*] treats "parasyphilis" as a purely syphilitic process comparable to other forms of tertiary syphilis elsewhere.

We now know that the central nervous system is very commonly infected, even in the absence of symptoms, in the earliest stages of the disease (11 per cent. in late *primary* syphilis).[†] Those tissues which are affected become "hypersensitive" to the virus, in the sense that they will respond to a further intoxication with an abnormally great reaction. Such increased susceptibility of the tissues is familiar in the ordinary gumma and in the lesions of tuberculosis. The spirochætes which caused the sensitizing infection in the early stages apparently die out after the secondary period has passed, except in certain areas where they persist as "rests." Such areas are in particular the wall of the aorta, the bones, and the central nervous system. If now the local balance between virus and immunity is upset, the spirochætes in a rest will multiply and give rise to a widespread reaction in the hypersensitive tissues. Should such a reaction occur around rests in the blood-vessels of the central nervous system, an interstitial lesion will result; but if the rest happens to lie among the nerve tissues, the lesion will also be parenchymatous. The parenchymatous cells will react to the intoxication, but, having no aptitude for repair, will succumb and degenerate. The lost function will then account for the symptoms of the disease. Such an explanation of the origin of parasyphilis is closely comparable with the results of a parenchymatous lesion of the testis—irreparable loss of function.

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[*] "A Comparison of the Lesions of Syphilis and Parasyphilis, together with evidence in favour of the identity of these two conditions," *Brain*, 1914, xxxvii. 141.

[†] P. Fildes, R. J. S. Parnell, and H. B. Maitland. 'Unsuspected Involvement of the Central Nervous System in Syphilis," Med. Res. Council, *Spec. Rept. Series*, No. 45. 1920.

The peculiar distribution of the lesions of parenchymatous syphilis, namely the affections of the posterior columns in tabes and the picking out of particular areas of the brain in general paralysis of the insane, is probably accounted for by a special distribution of the spirochætes in the acute stage by the perineural lymphatics connecting the superficies of the body with the central nervous system. The distribution of the syphilitic lesions would thus follow the same course as the cord lesions found in cases of bedsore by Orr and Rows. A perineural inflammation spreading up to the cord is a well-known phenomenon in experimental rabbit syphilis in the early stages.

Thus, according to this hypothesis, the stages which lead up to parenchymatous syphilis may be summed up as follows :—(1) Primary infection, (2) sensitization of the nerve tissues, (3) recrudescence of spirochætal activity in hypersensitive tissues, (4) abnormally active response of the hypersensitive tissues, (5) degeneration of neurones.

CHAPTER XXXII

GENERAL PARALYSIS OF THE INSANE (DEMENTIA PARALYTICA)

Etiology.—Men are more liable to suffer from the disease than are women, and it most commonly begins between the ages of 35 and 55. A history of previous syphilis can be obtained in a large number of cases, and the results of the Wassermann test, together with the discovery of the presence of the spirochæte in the brain tissue (Plate 14), have thrown a flood of light on the causation of this disease.

Wassermann and Plaut obtained a positive reaction of the Wassermann test with serum in 99·5 per cent., and with spinal fluid in 95 per cent. of the cases, while Browning found it in 96 per cent.

The presence of the spirochæte in the brain of general paralytics was first demonstrated by Noguchi, whose observations were soon confirmed by Marie, Levaditi, Mott and others. The organisms are found in small foci, and are generally most plentiful in the frontal lobes. While general paralysis must be regarded as due to syphilis in the vast majority of cases, if not indeed in all, it is generally accepted that in certain circumstances other conditions, such as worry, alcoholism, and injury, may precipitate the onset or accelerate the course of the disease; and it has been aptly said that general paralysis is a disease of syphilization and civilization. Where any question of this kind assumes a medico-legal aspect, the history and course of the disease in the particular instance must be carefully considered and the case judged on its own merits.

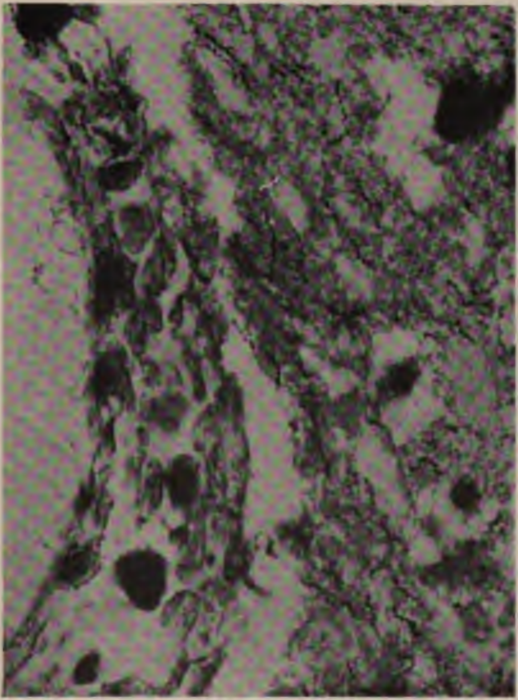

PLATE 14.—Spirochætes in the brain, near small vessel, in a case of general paralysis of the insane. × 1450. (*Sir F. W. Mott.*)

In some cases the onset occurs within a few years of infection, in others it may be delayed as long as twenty or twenty-five years, but the average interval between the time of infection and the onset of symptoms is about ten or twelve years. As already stated, the interstitial syphilitic lesions of the brain often arise within the first two years after infection—a fact which, in the opinion of Mott, suggests that the specific organism of general paralysis may have become in some way modified. The views concerning the paths by which the infection reaches the nervous system and the course which the degenerative processes take have been discussed in the previous chapter.

Juvenile general paralysis.—In a small percentage of the cases the disease arises in young people at about or soon after the age of puberty, and of these congenital syphilis is considered to be the cause.

Pathology.—To the naked eye, atrophy of the cerebral convolutions, especially those of the frontal lobes, and thickened and adherent membranes, are among the principal changes. The tendency for the frontal lobes to suffer out of proportion to the rest of the brain is interesting, and according to Mott it may perhaps be accounted for by the anatomical arrangement of the veins, which tends to produce a venous congestion of the circulation in this area. With the microscope, degeneration of cells and tracts of fibres connecting various parts of the cortex with one another (association fibres) can be demonstrated. The cortical cells, especially the Betz cells of the motor area, are swollen and altered in shape, and in the later stages of the disease many of them disappear, their place being taken by an overgrowth of neuroglia.

The vascularity of the cortex is increased, and new capillary formation can be seen. The endothelial cells of the capillary walls proliferate, and the perivascular spaces are filled with lymphocytes and plasma cells. The origin of the plasma cells is doubtful, but their presence is very characteristic of general paralysis.

In addition there are "mast" cells and "rod" cells to be seen.

Symptoms.—The first symptoms are usually those connected with the mind. The patient becomes irritable, unstable, and alternately unduly depressed and exalted. Early mental changes vary in character in different individuals: in some patients depression is a feature; in others exaltation is present. Convulsive seizures, though commoner in the later stages, are sometimes among the first symptoms to be noticed.

Forgetfulness is especially apt to be noticed as an early symptom when the patient is a business man, for naturally any mistakes are soon liable to be found out; but where the patient has not a responsible position, loss of memory, although perhaps present, is more likely to be overlooked. The same is true of manual work; the last and most delicate acquirements are the first to decline, on account of failure of the attention and of the delicate coördination that are required for their performance.

When the patient is brought for examination there are frequently definite signs which, taken with the mental condition, make the nature of the case clear. Of these the most common are inequality of the *pupils* with failure to react to light, a "slurring" *speech*, and *tremors* of the lips, tongue, and hands.

The pupils are like those of locomotor ataxy (Argyll-Robertson), and contract during accommodation but not to light. Their inequality is often marked, and they have often ceased to be circular, and exhibit an "angular" appearance. There is no nystagmus, and in the early stages the optic discs do not usually show any change, although later on they may undergo atrophy.

The speech is generally tested by the ability to say such words as "artillery," "constitutional," and "constabulary." There is a great tendency to elide syllables, a tendency which is also noticed in the writing (Fig. 73). In talking there is a fine tremor of the lips and a general over-action of all the face muscles; a fine tremor may also be observed in the tongue.

The hands tremble, and there is loss of accuracy in movements requiring fine coördination, as can often be demonstrated by asking the patient to button up his coat.

The tremor, incoördination, and general failure of intellect are often all made manifest in the writing (Fig. 73).

The knee-jerks may be absent or increased, and in the latter event they are often unequal; the plantars may give an extensor response.

Diagnosis.—General paralysis of the insane is often seen by the neurologist during its early stages, when it is most important to arrive, if possible, at an accurate diagnosis. In the early stages, care must be taken to avoid confusion with *neurasthenia*. Occasionally it may at first be impossible to make a certain diagnosis, but usually there is a difference between the simple fatigue symptoms of neurasthenia and the mental degeneration of general paralysis; and with the development of physical signs the diagnosis becomes clear. The neurasthenic worries about himself, but in general paralysis it is the friends who are often first worried on account of the mental symptoms which change the man from his former self into an irritable, careless, and strange being.

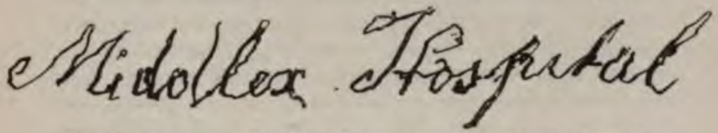

Fig. 73.—Specimen of writing from a case of general paralysis.

Interstitial forms of cerebral syphilis are often very difficult to distinguish from general paralysis; and the methods which may assist in the differentiation have already been discussed (p. 268). That the two should be distinguished is most important, for while the prognosis of the meningo-vascular form, if it be adequately treated, is comparatively good, that of general paralysis is almost hopeless.

This disease may also bear some resemblance in its early stages to *disseminated sclerosis*, the chief symptoms common to both being tremors, alteration in speech, and increase of deep reflexes; but it must be remembered that the knee-jerks are by no means always increased in general paralysis—they may be diminished, unequal, or absent. The tremor of general paralysis is finer than that of disseminated sclerosis, and it is, moreover, especially marked in the face, tongue, and lips. The speech, too, when well developed, differs in the two conditions: in general paralysis it is hesitating and syllables are dropped or telescoped into one another; while in disseminated sclerosis it is jerky, staccato, or scanning—characters which are rather difficult to describe, but which are distinctive when heard.

Among other conditions which may sometimes closely simulate general paralysis are some phases of mental disorder due to *chronic alcoholism*. *Tumours* of the *frontal lobes* with mental symptoms and tremors may also bear some resemblance to it, but the presence of optic neuritis and the absence of Argyll-Robertson pupils help to serve as distinguishing features.

Prognosis.—The prognosis is always bad, and the patients, with the exception of occasional remissions, tend to go steadily downhill. *Fits*, epileptiform or apoplectiform, are often a marked feature, and each attack generally leaves the patient both physically and mentally worse than he was before. As the mind deteriorates the physical weakness increases, until at last the patient arrives at the state which justifies the use of the term "general paralysis."

Treatment.—That the treatment of general paralysis is not usually followed by satisfactory results is due chiefly, it would appear, to the difficulty which the drugs encounter in reaching the spirochætes.

It has long been known that no good results can be expected from giving mercury, and at present but little, if any, more benefit can be obtained from salvarsan. Various methods of bringing the latter drug into closer contact with the spirochætes have been tried, the chief of which is that proposed by Swift of injecting immunized serum into the cerebro-spinal fluid, into which salvarsan itself cannot, of course, be introduced. Various modifications of this method have been tried, e.g. subdural injections and injections into the lateral ventricles, but unfortunately up to the present with little definite success.

Treatment otherwise consists in placing the patient under proper care and control, giving attention to the general health, and trying to relieve individual symptoms as they may arise.

CHAPTER XXXIII

TABES DORSALIS (LOCOMOTOR ATAXY)

Etiology.—The symptoms of locomotor ataxy are due principally to degeneration of the lowest sensory neurones. But although the degeneration mainly attacks the sensory neurones, some signs of its extension to those of motor function also are often seen, more especially in connection with the ocular nerves, and occasionally, though less frequently, in connection with motor nerves in other parts of the body.

The disease is found in men about ten times as often as in women, and the onset in most cases occurs somewhere between the ages of 20 and 50. The time at which symptoms appear after the primary infection is very variable and may be as long as twenty-five years or more, but the average is about ten or twelve years. Occasionally patients with the disease are seen while in their teens, and to these cases the term *juvenile tabes* is applied. Though it is possible that injury, alcoholism, worry and other debilitating influences may act as contributory causes, the application of the Wassermann test and the discovery of spirochætes in the central nervous system have placed the dependence of locomotor ataxy upon a previous attack of syphilis beyond all reasonable doubt, and have, moreover, demonstrated a direct relationship between the two conditions; for while formerly it was thought that syphilis acted indirectly by lowering the vitality of the neurones, it is now known that the action is directly due to the activity of the spirochætes, and that tabes dorsalis is to be regarded as one of the parenchymatous forms of syphilis of the nervous system.

The reason for the definitely selective action of the toxin on the lowest sensory neurones has been much debated. It is well known that poisons like lead, diphtheria, alcohol, and arsenic all exercise a selective action on certain groups of fibres, and it may be that the poison which causes tabes acts in a similar manner.

One view is, that the poison injures the cell bodies from which the fibres arise and receive their nutrition. In this instance the cell bodies are situated in the posterior root ganglia, and changes in them have been described, although they do not appear to be demonstrable in every case. Moreover, if failure of the nutrition of these cells causes degeneration of the central process of the T-shaped fibres which spring from them, one would expect the peripheral process to degenerate as well, but this does not by any means always occur. Changes have, however, been described in the muscle spindles in which the periphery of some of the nerves terminates, and it may be that these are the first indication of a peripheral degeneration (Batten).

There are anatomical reasons which seem to make the fibres of the posterior nerve roots comparatively less resistant. There is, for example, some slight natural constriction of the roots as they pass through the pia mater. This has been thought to lower their resisting powers, and some neurologists consider that the degeneration is produced by a thickening of the spinal membranes which compresses the roots as they pass through the pia mater, i.e. at the point at which they are naturally rather narrowed, and that this thickening is caused by a tertiary syphilitic process. That some thickening of the membranes is frequently present is undisputed, but it is difficult to conceive it as the main cause of the degeneration. If it were so one would expect the anterior roots to suffer more frequently; and, further, there is also the difficulty of accounting for the ocular and other cranial nerve symptoms on this hypothesis.

The part which the lymphatics play in the selective nature of the degeneration is probably very important, and the hypothesis concerning the sensitization of the posterior columns has already been discussed in the section dealing with the general aspect of parenchymatous syphilis (pp. 276, 277).

Orr and Rows * have published the results of some important researches on this subject, which show that "in peripheral nerves, spinal roots, and cranial nerves there is a constant stream of lymph ascending towards the central nervous system, whose main current lies in the inner meshes or lymph spaces of the fibrous perineural sheath." Toxins reaching the spinal cord and brain by this channel are particularly apt to affect the nerves where they have lost their neurilemma sheath, which spot, Orr and Rows point out, is not quite identical with the place at which the nerves undergo a slight constriction as they pass into the cord. In this way the distribution of the lymph may be the determining cause of the locality of the degeneration, and it is possible, as suggested by Mott, that the immediate cause of the degeneration is produced not always by the organisms themselves, but by a proteolytic ferment which arises outside the cord and then passes up to the cord by the lymphatics of the posterior roots. Thus the steps of the process would be: (1) focus of infection; (2) production of proteolytic ferment; (3) circulation of ferment in the lymphatics of the posterior roots; (4) degeneration of the nerve fibres.

Pathology.—It is generally agreed that degeneration of the nerve fibres is the primary event, and that the overgrowth of neuroglia is secondary, and this view receives confirmation from the fact that the only degenerated fibres traceable in the cord during the early stages of the disease are those which are continuous with the roots (i.e. exogenous fibres), and not those which pass from one part of the cord to another (i.e. endogenous fibres). Were the primary condition an overgrowth of neuroglia, no such distinction would be shown between the different sets of fibres.

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* *Journ. of Neurol. and Psychiatry*, May, 1907.

Of these fibres from the posterior roots which are so prone to degenerate, some proceed up the posterior columns, some complete the reflex arcs by communicating with the cells of the anterior horns, and some terminate in Clarke's column, from which fibres arise to pass up the direct cerebellar tracts (Fig. 74, A, B, and C), while others end in the cells of the grey matter.

Degeneration is most prominently marked in connection with those fibres which pass up the posterior columns of the cord, and if the course of a single degenerated fibre be traced it will be observed first to pass up the posterior external column (Burdach), subsequently to find its way into the posterior internal column (Goll), and then to pass up to the medulla, where it terminates.

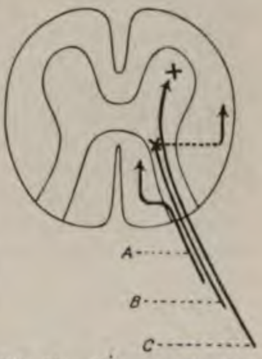

Fig. 74. — Diagram showing the main destinations of fibres of the posterior nerve roots. (*See* text.)

Thus a section of the cord shows degeneration of the posterior columns, and, since the roots in the lower part of the cord are generally the most affected, the degeneration is there most dense in the posterior external columns, while it gradually becomes more pronounced in the posterior internal columns as it ascends the cord.

Mechanism by which symptoms are produced.—The symptoms of locomotor ataxy being the result of degeneration of fibres in the posterior nerve roots, consideration of the functions of these fibres and of the consequences following their destruction will give a good idea of the different symptoms that occur.

A posterior nerve root contains (1) fibres carrying impulses for sensations of touch, pain, and temperature;

(2) fibres along which impressions pass from the muscles, ligaments, and joints; and (3) fibres which complete the reflex arcs by communicating with the cells in the anterior horns.

(1) Failure of nutrition of the fibres which convey impressions of touch, pain, and temperature results in anæsthesia over the areas of skin corresponding to the particular roots affected; but since all the fibres in the root are not damaged to an equal extent, until the root is entirely destroyed the anæsthesia occurs in patches, some part of a given root-area being more anæsthetic than others.

Further, one form of sensation may suffer more than another, so that the patches of anæsthesia to touch, pain, and temperature do not necessarily coincide.

This degeneration is accompanied by signs of irritation of the nerves which takes the form of "lightning pains" and visceral crises.

The loss of recognition of sensory impulses is closely associated with modifications of nutrition in the skin, bones, and joints, constituting the so-called trophic changes.

(2) The degeneration of fibres which should convey impulses from the muscles, ligaments, and joints is accompanied by incoördination and a failure to recognize the relative positions in which various parts of the limbs are placed (i.e. loss of sense of position). The power of distinguishing the forms of objects is also frequently diminished (astereognosis).

(3) Degeneration of the fibres which communicate with the cells of the anterior horns leads to loss of reflexes and to loss of tone of the muscles. The impulses which constantly flow into the cord through the posterior roots exercise a definite influence upon those flowing out by the anterior roots, and if they are cut off either by experiment or by disease as they are in the case of locomotor ataxy, the tone or elasticity of the muscles of the corresponding arc is diminished, and the condition known as hypotonus arises (Fig. 75).

If these principles are applied to different levels of the cord the mechanism of the main symptoms is clearly demonstrated.

Thus from degeneration of the lumbar and sacral roots there arise pains in the legs, patches of anæsthesia, numbness and a feeling as though walking on cotton-wool, trophic symptoms, incoördination, loss of the knee-jerks and tendo-Achillis jerks, impairment of the action of the sphincter reflexes, and hypotonus.

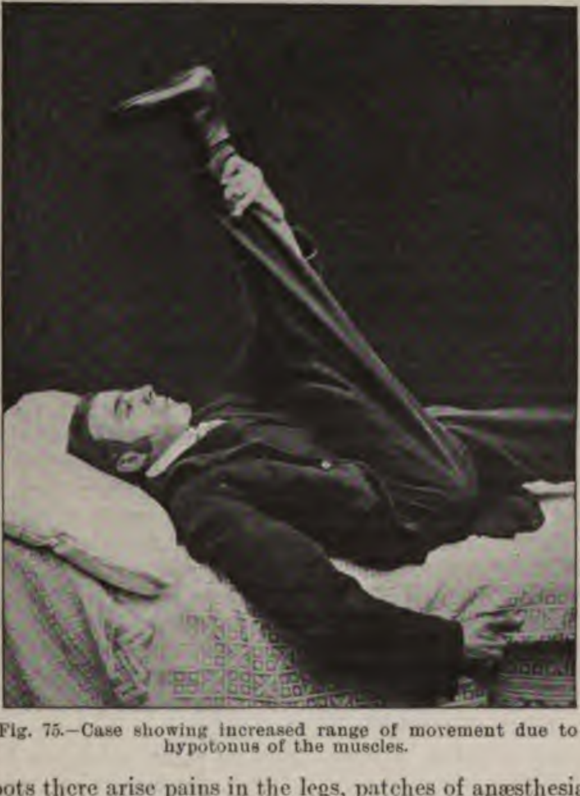

Fig. 75.—Case showing increased range of movement due to hypotonus of the muscles.

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In the dorsal region the degenerating fibres are apt to cause girdle pains and a zone of altered sensation in the corresponding course of distribution of the roots.

Higher still—in the cervical region—there are signs in the arms corresponding in character to those already enumerated for the legs; and at a still higher level the reflex arc for light is broken and the optic nerve atrophies.

Such, then, is the general mode of production of the sensory symptoms of locomotor ataxy, over and above which there may be many others referable to alterations in the motor neurones.

General clinical course.—It will be clear from what has already been said that there can be no constancy concerning the order in which the different symptoms make their appearance.

The *onset* is generally associated with *pains*, which, as might be expected, may be felt for a long time, even for years, before the attention of the patient is called to any other symptom, though it must be admitted that a systematic examination would probably often reveal the existence of other signs at an earlier date than they are otherwise recognized. The pains are of a lightning or shooting character, and vary greatly in their intensity and duration, often disappearing for weeks together, to return subsequently for several days at a time. Associated with these there may be hyperæsthesia of such intensity that the patient cannot bear the slightest touch. At first the pains are almost invariably attributed to neuralgia or rheumatism, or, if they remain for a time confined to the course of a particular nerve, they are liable to be attributed to a localized neuritis, e.g. of the sciatic or ulnar nerve.

In addition to these lightning pains which are felt in the more superficial structures, there may be deeper "boring" pains in the bones or other organs, which often remain for a long time localized to one spot. Cramps in the muscles are also sometimes a prominent feature.

Since the lumbar and the sacral roots are generally among the first to degenerate, the *knee-jerks* and the *tendo-Achillis jerks* are, as a rule, lost or much diminished quite early in the course of the disease, and at the same time changes are very frequently occurring at the other end of the system which result in loss of the *light reflex.*

Thus the combination of symptoms most commonly found early in the disease consists of lightning pains, loss of knee-jerks, and the Argyll-Robertson pupils, i.e. pupils which contract during accommodation but do not react to light, and which may be pin-point or unequal in size.

In addition, if carefully looked for, there is a likelihood of some degree of *ataxy* being detected in the lower extremities; but other symptoms often exist for a long time before ataxy is noticed, so much so that a "preataxic stage" of the disease is clinically recognized.

Another early, and not infrequently the first, symptom is that of *primary atrophy of the optic nerves.*

The clinical course of the disease may best be described by following the development of the chief symptoms (Fig. 76) in a typical case.

If the patient comes complaining of "rheumatic" pains, which on inquiry prove to be of a lightning character, and examination shows loss of knee-jerks and the presence of the Argyll-Robertson pupil, the diagnosis of tabes in the "preataxic" stage will probably be made.

In such a case other signs will in all probability gradually show themselves, although in favourable circumstances the progress of the disease may be very slow or may even become arrested.

If degeneration progresses, the patient's attention will sooner or later be attracted by his unsteadiness both when walking and standing, this unsteadiness being greater in the dark or on such occasions as he may have his eyes shut, e.g. when washing his face. Before, however, the patient recognizes any difficulty in this direction it can generally be discovered by making him stand with his feet together and his eyes closed (Romberg's sign), or by asking him to try to execute such balancing

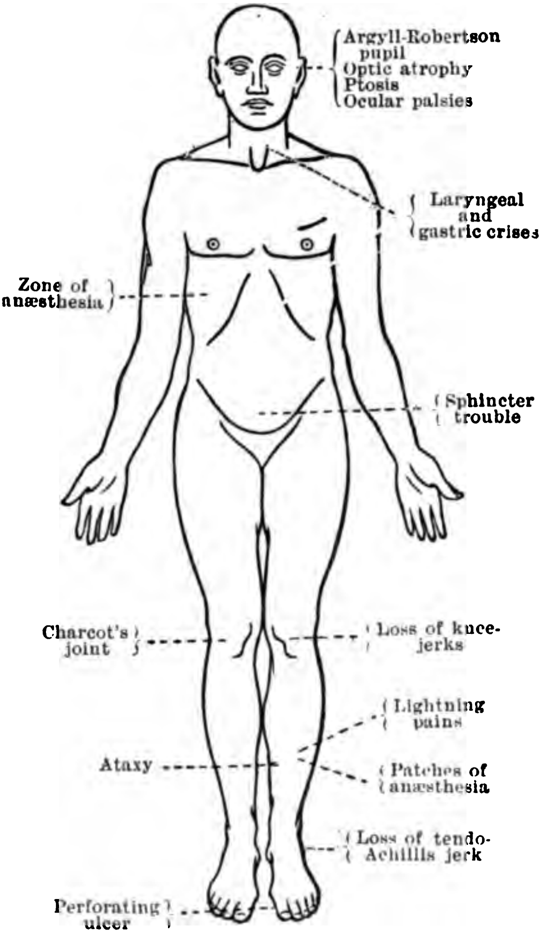

Fig. 76.- Scheme of the principal symptoms of tabes dorsalis.

movements as are necessary for walking with the heel of one foot placed at each step in front of the toes of the other—a very delicate test if performed with the eyes closed.

Various defects in the sensations of touch, pain, and temperature will be found in irregular patches corresponding to the areas supplied by the degenerating nerve roots, and the sole of the foot is a particularly likely place to find such patches. The areas of loss to one kind of sensation do not necessarily correspond to those of another, as is only to be expected when it is remembered that the separate nerve fibres are degenerating independently of one another.

Another sensory disturbance often found quite early in the disease is a zone of analgesia or anæsthesia round the chest or abdomen, indicating that degeneration is proceeding in some of the dorsal roots.

Sexual power is usually greatly diminished or lost by the time the disease is fully developed, but during the early stages it may be temporarily increased.

If the case be progressive, the ataxy now increases in degree, until at last the patient may be quite unable to walk or even to stand. Before, however, this stage is reached many symptoms may or may not develop incidentally in an order which cannot possibly be prognosticated, for, as already insisted upon, almost any one of all the possible symptoms occurring during the course of the disease may be the first to attract attention.

With this general sketch of the disease it is now necessary to consider certain of the main symptoms in some detail.

Trophic disturbances. *Perforating ulcers.*—Perforating ulcers are characterized by the formation of crateriform indolent sores of various depths, which make but little attempt to heal. (Fig. 77.) The skin at their edges is usually thickened and raised to form a dense callosity, which often exists as a corn before any ulceration takes place.

The ulcers, which tend to assume a somewhat conical shape, are generally painless, and frequently extend right down to the bone.

Their favourite position is on the fore part of the sole of the foot, but sometimes they appear on the upper surface of the toes; they show no disposition whatever to occur on the heel.

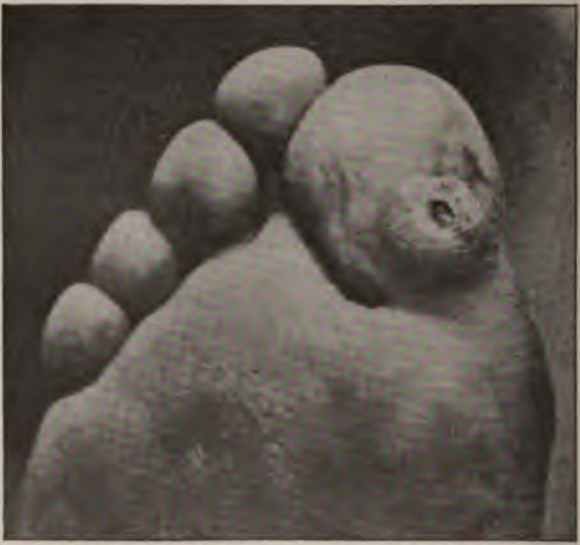

Fig. 77.—Perforating ulcer in a case of tabes dorsalis.

The underlying factor in the formation of these ulcers is undoubtedly an alteration in the nutrition of the affected parts consequent upon the destruction of sensory fibres, while pressure and injury presumably take a share in the causation. In some cases, as suggested by Tourette, the ulceration may possibly be secondary to diseased bones and joints, and may be in fact a fistula. Under favourable conditions, the most important of which is rest, an ulcer often heals spontaneously, but sometimes first requires such stimulation as is obtained by scraping or by exposure to the X-rays, the latter mode of treatment appearing in some cases to act very favourably.

Osteo-arthropathies.—Changes in the bones and the joints have next to be considered—the so-called osteo-arthropathies.

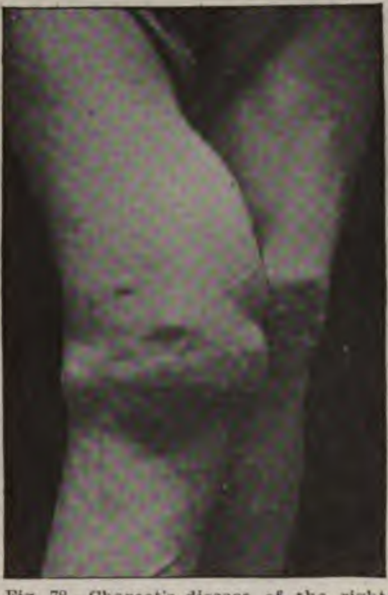

Fig. 78.—Charcot's disease of the right knee.

The condition known as *Charcot's joint* may occur at practically any period of the disease, and is characterized by a painless and often rapid swelling due to the effusion of fluid, which, after being absorbed, often leaves the joint in a flail-like condition, due partly to the laxity and softening of the ligaments and partly to changes in the articular surfaces of the bones; these last-mentioned changes may all occur without any definite degree of effusion. The knee-, elbow-, hip-, and shoulder-joint are among those which most commonly suffer, while another common situation, perhaps not so often recognized, is that of the big-toe joint. (Figs. 78, 79.)

The essential process in all these joints is one of atrophy, which is sometimes accompanied by irregular bony outgrowths similar to those associated with rheumatoid arthritis. This latter form, to which the term hypertrophic is sometimes applied, appears to occur more often in the knee-joint than elsewhere, while the shoulder and hip are more likely to show signs of advanced atrophy, as evidenced by dislocation of the heads of the bones.

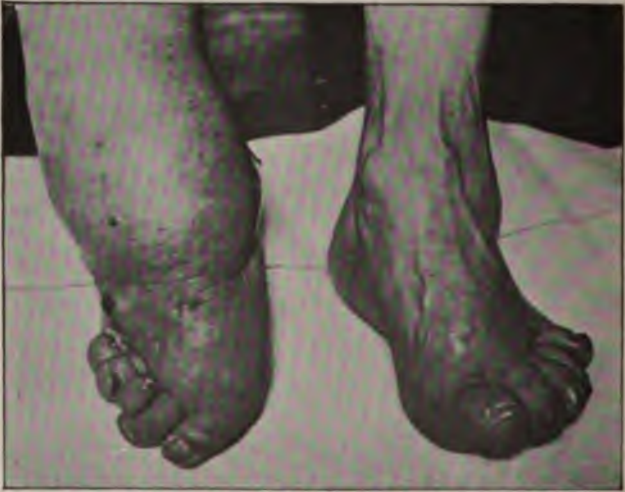

Fig. 79.—Charcot's disease of the right ankle-joint associated with atrophic changes in the bones of the foot.

The exact mechanism by which these joint troubles are produced is obscure. The influence of traumatism as a direct cause can be largely excluded by their frequent occurrence in the preataxic stage of the disease, and the possibility of their being of syphilitic origin is negatived by the fact that changes of practically the same nature are to be found in syringomyelia, concerning which there appears to be no question of the influence of syphilis.

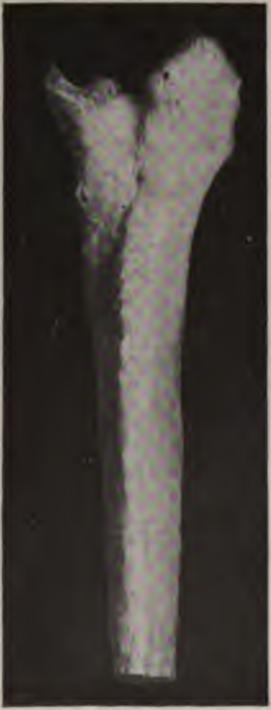

Fig. 80.—Atrophy of the head of the humerus associated with a "Charcot's joint." (*Middlesex Hospital Museum.*)

The bones.—In locomotor ataxy the proportion of the lime salts in the bones is frequently reduced, hence

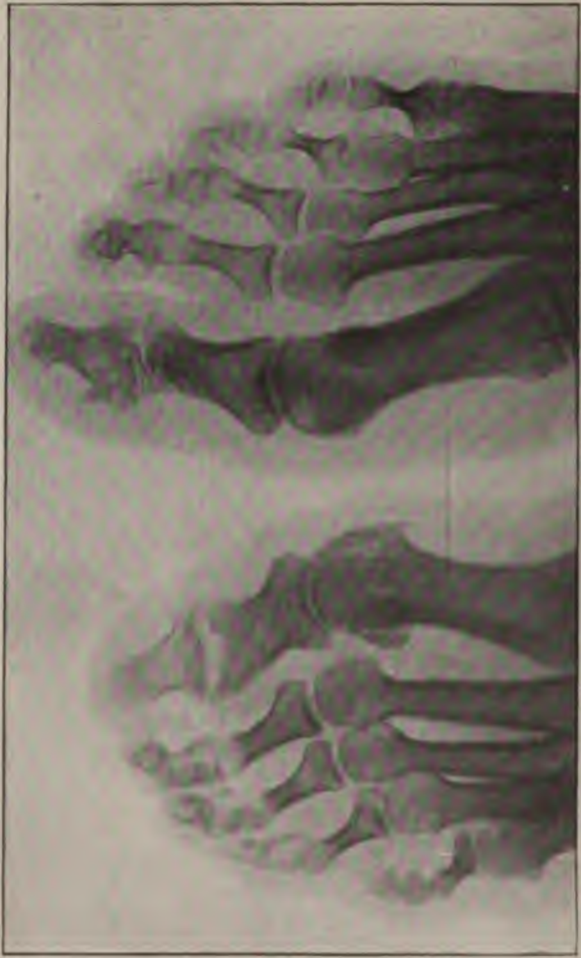

Fig. 81.—Skiagram showing atrophy of the bones of the left foot. Note the shortening of the great toe.

the bones become soft and brittle, and "spontaneous" fractures (i.e. fractures brought about by a very slight amount of violence) arise.

The atrophy of bones (Fig. 80), especially those of the feet (Figs. 81, 82), is often obvious during life. The big toe is very frequently shortened, owing, as shown by the X-rays, to wasting of its bones. In the feet and, more rarely, in the vertebræ the bones and joints are apt to show changes at the same time.

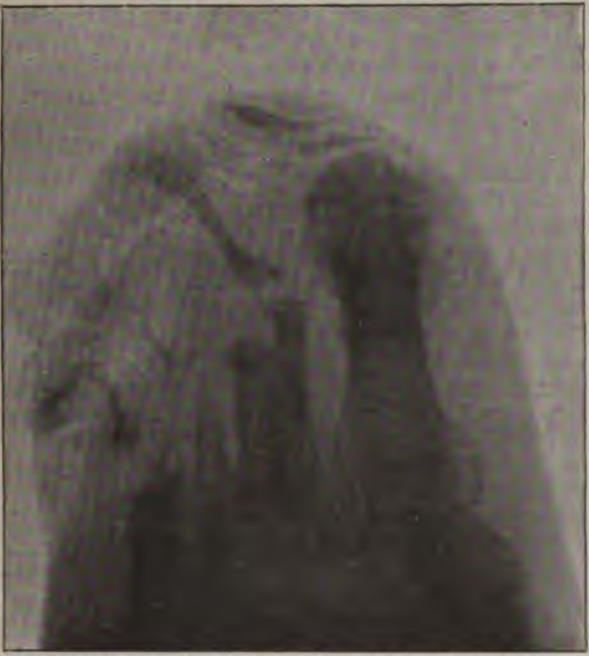

Fig. 82.—Skiagram showing very advanced atrophy of the bones of the foot. (*From a case under the care of Mr. T. H. Kellock.*)

The crises.—Paroxysmal disturbances of function (known as crises) are apt to occur in connection with the various viscera. In their production they appear to be analogous to the lightning pains, and for the most part they probably depend upon degenerative changes in the sympathetic system.

Gastric crises.—These form the commonest variety of crises. Every few weeks the patient has attacks of intense pain and sickness, which for the time being entirely incapacitate him from work. The duration of these attacks varies from a few days to a week or more, and in the intervals there may be no symptoms whatever.

Wilfred Harris has observed the occasional occurrence of epileptiform attacks during the gastric crises, and considers that there is some causal relationship between the two conditions.

Laryngeal crises.—In these the patient gets signs of spasm of the laryngeal muscles, accompanied by a barking and crowing cough which in some ways resembles whooping-cough.

Other organs subject to crises are the *heart*, *bladder*, *rectum*, and *kidneys*.

Ocular symptoms.—The *Argyll-Robertson pupil* is one of the commonest symptoms of tabes dorsalis. Its characteristic is loss of reaction to light, while contraction during accommodation remains. (Fig. 83.) The pupils are generally very small (pin-point), often unequal, and frequently show some loss of that perfect rotundity by which they are distinguished in health.

The Argyll-Robertson pupil is often the earliest sign of tabes, and it may occasionally be observed to exist for a time in one eye only. When the degeneration is more extensive the contraction during accommodation may also be absent.

The lesion causing the loss of light reflex in the Argyll-Robertson pupil is possibly situated in the fibres which complete the reflex arc by joining the optic nerve to the nucleus of the 3rd nerve (Fig. 83). In a case in which there is no question of optic atrophy, it is obvious that the fibres of the optic nerve are healthy, and in like manner the ability of the pupil to contract in association with accommodation shows that impulses can pass freely along the fibres of the 3rd nerve. The inference, therefore, is that the degeneration in such a case has taken place in the fibres between the two nerves.

Another view is that the Argyll-Robertson pupil is caused by degeneration of the ciliary ganglion.

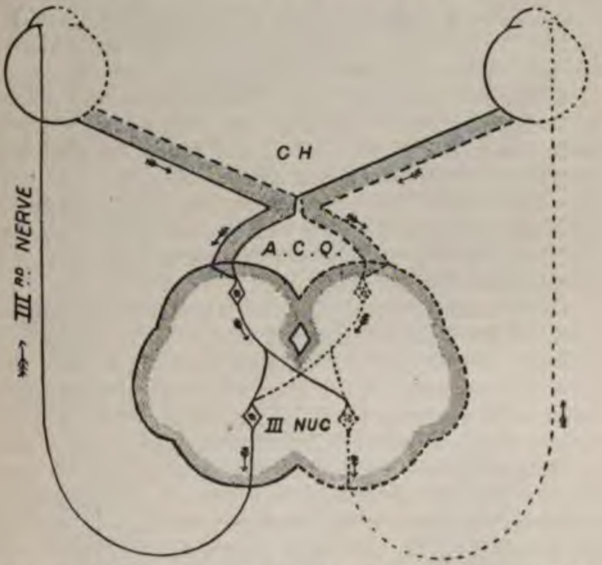

Fig. 83.—Diagram of the pupil reflex to light. (*Wilfred Harris.*)

Optic atrophy.—A primary atrophy of the optic nerve is apt to occur in the course of locomotor ataxy, and sometimes failure of sight from this cause is the first symptom to attract the patient's attention. The disc assumes a pearly-white appearance, and the vessels running over it are relatively small. The rate at which sight is lost varies greatly, and it may be some three, four, five, or more years before anything like complete blindness supervenes.

Paresis of muscles.—Paresis of the ocular muscles indicates a lesion of the motor neurones, and it is in this region that the motor symptoms in tabes are most commonly found.

Any of the ocular muscles may be affected, but the external rectus is the one most prone to suffer. Squint and diplopia are the result, but as a rule the paresis is only of temporary duration, and it is common for patients to complain from time to time of double vision, which completely passes away in the intervals.

Much less common, but more important, are the permanent paralyses in which all the branches of the 3rd nerve are liable to be affected, and which are probably due to degeneration of the motor nuclei.

Ptosis is sometimes temporary, thus resembling the transient paralysis of other ocular muscles, but at other times it may be permanent and be part of a complete paralysis of the 3rd nerve (Fig. 84).

Other cranial nerves.—No cranial nerves are exempt from the possibility of degeneration; the olfactory and auditory nerves may suffer, and sometimes there are signs of degeneration of the nuclei in the medulla. Anæsthesia and pains not infrequently occur over the regions supplied by the 5th nerve.

Diagnosis.—In considering the diagnosis of locomotor ataxy it is important to remember the possibility of the signs appearing in almost any order. In one case the pains may be the most prominent symptom; in another a joint may be the chief cause of anxiety; in a third it may be the eyes that first give rise to trouble; and in yet another there may be difficulty in exercising full command over the bladder. It is most necessary in diagnosing tabes to remember that undue stress must not be laid upon the absence of one or more signs that ought to be present in a "typical" case. For instance, because the knee-jerk is usually lost by the time most patients come for diagnosis, its presence, and sometimes even its increase, must not be allowed to negative the possibility of tabes if other signs point to such a conclusion. When the roots are affected in such a variable order as they are in tabes, it is no wonder if those upon which the presence of the knee-jerks depends are not always among the first to degenerate.

The presence of Argyll-Robertson pupils in association with any other suspicious symptom—e.g. lightning pains, bladder trouble, or a swollen joint of doubtful nature—is practically pathognomonic of tabes, provided that the onset of general paralysis of the insane can be excluded.

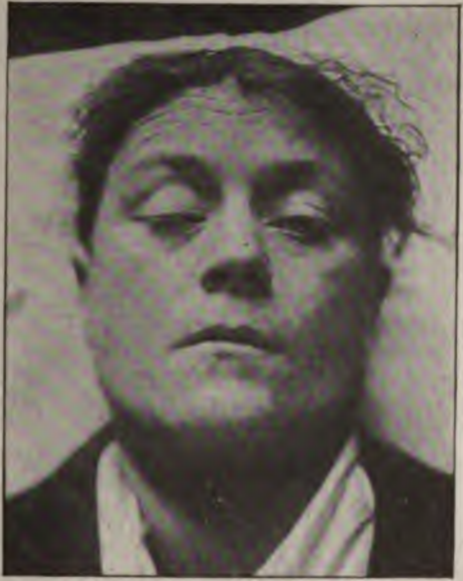

Fig. 84.—Case of tabetic ptosis, showing the compensatory contraction of the occipito-frontalis.

It occasionally happens that, during the routine examination of patients, Argyll-Robertson pupils are discovered as isolated signs and are associated with no other detectable morbid conditions whatever. The question naturally then arises as to what interpretation should be put upon such a case. On the whole it would seem that the most reasonable attitude to adopt in such cases is to look upon the sign as evidence of local degeneration, probably of a parenchymatous syphilitic nature; but experience shows that, although its presence is very suspicious, it may nevertheless exist for an indefinite time without signs of degeneration of the nervous system appearing elsewhere.

Peripheral neuritis.—Cases of peripheral neuritis in which sensory symptoms are prominent sometimes resemble tabes in many respects, for common to the two disorders may be pains, loss of knee-jerks, and incoördination, and occasionally the similarity is increased by the presence of sores on the feet, which may be mistaken for "perforating ulcers." The closest similarity seems to occur in some of the cases of neuritis due to alcohol and to diabetes, but a systematic examination generally removes the doubt by revealing positive signs of one or other disease. An Argyll-Robertson pupil, for instance, would be conclusive evidence in favour of tabes, while loss of power and electrical changes would strongly support the supposition of neuritis.

Friedreich's disease has several symptoms in common with tabes—e.g. loss of tendon reflexes and incoördination—but the age of onset and the family nature of the disease, together with the speech defects, nodding movements, and nystagmus, usually serve to distinguish it, though, as far as age is concerned, it must be remembered that cases of juvenile tabes may occur.

Ataxic paraplegia.—The combination of spasticity with incoördination, together with the increased knee-jerks and the absence of eye symptoms, will serve to distinguish ataxic paraplegia from tabes.

Cerebellar disease.—The ataxy of cerebellar disease does not usually become distinctly worse when the eyes are closed.

If there is a tumour the history and symptoms will not be suggestive of tabes, and if the ataxy is due to cerebellar deficiency the history of the case will enable tabes to be excluded.

Disseminated sclerosis.—Disseminated sclerosis appears in so many forms that in some of them it may occasionally resemble tabes, but, even in those instances where confusion is likely to arise, exaggerated knee-jerks (loss of knee-jerks is very uncommon in disseminated sclerosis), alteration in speech, nystagmus, and tremors on movement are generally present in some degree.

Interstitial spinal syphilis.—Syphilitic disease of the membranes, causing secondary changes in the posterior roots, combined as it may be with thrombosis of vessels in the posterior columns of the cord, may give rise to signs which are in some ways almost indistinguishable from those of tabes.

Pain in the back, loss of power (of flaccid type, due to implication of anterior roots, or of spastic type, due to extension of the thrombotic processes into the lateral columns), and the variability of the symptoms are among the distinguishing features. The presence of co-existing signs of interstitial cerebral syphilis often assists in making the diagnosis, but it must be remembered that parenchymatous and interstitial disease of the brain and cord may exist at the same time.

Prognosis.—Probably in no other chronic disease of the spinal cord does degeneration vary more in its rate of progress than it does in tabes.

After the appearance of the first symptoms, many years often elapse before there are any signs of ataxy. The symptoms may cease to progress at various stages of the disease, and in a certain number of cases some definite improvement occurs. In these circumstances it will be obvious how important it is to try to form an estimate of the probable future of every case in order to be in a position to give some practical advice regarding the best course for the patient to pursue.

This, as far as is possible, may be accomplished by paying attention to the patient's age and circumstances, the rate at which the degeneration has progressed, and the position of the first signs of degeneration.

It has been observed that the course of tabes is, as a rule, much slower when it begins in elderly people of 50 or 60 than when it attacks those of younger years, e.g. from 25 to 30. In these latter the degeneration often proceeds very rapidly.

The circumstances of the patient must be taken into account in forming a prognosis, since those who have money and leisure are in a better position to combat the disease than those who are obliged to continue their daily work.

If at the time of observation the patient gives a history of tabetic symptoms existing over a period of some years, and examination shows that the extent of the degenerative processes is still very limited, it is reasonable to infer, with certain reservations, that the changes will continue to proceed slowly.

In this connection Sir William Gowers directed attention to a group of cases in which, though lightning pains have existed for a number of years, there is no loss of knee-jerks and no sign of ataxy. According to Gowers, these should be considered as a special variety of tabes depending upon some peculiarity of the toxin rather than ordinary cases in which development of the disease has been abnormally slow. The future of such cases appears to be comparatively favourable, and until the knee-jerks are lost no ataxy is likely to arise.

It is also well known that patients who develop optic atrophy early often remain for a long time without any signs of ataxy.

In predicting a slow degeneration for the future on the strength of one that has been slow in the past, it must not be forgotten that very rapid increase in the intensity of symptoms is sometimes brought about by shock, illness, exposure to cold, or injuries.

Treatment.—The difficulty in obtaining favourable results from the administration of mercury and salvarsan in cases of parenchymatous syphilis has already been emphasized, and up to the present there is little evidence of any striking benefit having been produced by these methods of treatment. Preparations of salvarsan given intravenously or intrathecally in the form of immunized serum appear in some instances to have been followed by relief of lightning pains and some general improvement, but there does not seem to be any conclusive evidence that the progress of the disease can be permanently arrested by this method. It appears to be likely, however, that more benefit may be obtained by systematic treatment in the case of tabes than in general paralysis, since the progress of tabes is so much slower, and frequently becomes more or less stationary; and it is usually considered advisable for the patient to undergo a course of antisyphilitic treatment from time to time as a precautionary measure against further extension of the disease. If the hypothesis mentioned above (p. 286), that in some cases the mischief is produced by a proteolytic ferment which has its origin externally to the cord, should prove to be correct, it would seem that the antidote should have a better chance of reaching the focus of infection than in cases where the trouble is due to activity of the spirochæte within the cord; hence, for the present it will be well to keep an open mind on the effects of treatment in cases that have been carefully selected as likely to receive benefit.

Apart from antisyphilitic remedies, reliance must be placed on increasing the resisting powers of the patient by improving his nutrition, which can best be done by prescribing suitable tonics, with a proper amount of food and rest.

Of tonics, the author places most reliance on iron, and it seems reasonable to suppose that an improvement in the quality of the blood will be followed by an improvement in the nutrition of the nerve fibres. The perchloride, in doses of 10 to 15 minims three times a day, appears to answer best, but, if it disagrees, other preparations may be substituted. If it be thought advisable, arsenic may be given with the iron. Aluminium chloride was introduced into therapeutics by Sir William Gowers for its apparent usefulness in modifying the intensity and frequency of the lightning pains, and it also may be given in combination with the iron and arsenic in some such prescription as the following :—

℞ Tincturæ ferri perchloridi ...	Dl. 7 ...	♏xii
Liquoris arsenici hydrochlorici	Cl. 18 ...	♏iii
Aluminii chloridi	Dg. 2 ...	gr. iii
Spiritus chloroformi	Ml. 1 ...	♏xv
Aquam ... ... ad	Ml. 30 ...	℥i

Two tablespoonfuls to be taken three times a day after meals.

Strychnine is a drug which may also occasionally be given with benefit.

Plenty of nourishing food should be taken, for the nutrition of these patients is frequently poor, and, in many instances, cod-liver oil is useful.

Rest is an important factor in the course of treatment. The degeneration seemingly has a tendency to occur in those nerves whose functions are most hardly used, and to continue to exercise these nerves as much as before is only to court an extension of the degenerative processes.

Walking (for the disease generally begins in the roots at the lower part of the cord) should be slow, the distances should be short, and, whenever possible, driving should be substituted. Fatigue both of mind and body should be avoided as much as possible, and it is scarcely necessary to add that plenty of fresh air, within doors as well as without, is essential.

The various forms of treatment by electricity have, at most, nothing more than a passing tonic effect.

To restore the functions of those fibres which are already dead is, of course, impossible, but in suitable circumstances, and by careful attention to detail, there is no doubt that something can be done to retard the extension of degeneration.

It must also be recollected that in disease, just as in health, function is often a degree above or below what may be the average level for any particular person. Tabetic patients with some degree of optic atrophy can see much better on some days than others; so, also, those with ataxy can walk better on some days than others. To keep these patients at their highest possible level of health should be one of the objects of treatment.

Treatment of lightning pains.—To relieve the lightning pains of tabes is often a difficult matter. Among the most reliable drugs are phenacetin, antipyrin, antifebrin, and aspirin; sometimes success is obtained with one and sometimes with another, and occasionally a combination is very effective. They may be given conveniently in cachets, and, when the attack is very severe, Tourette has recommended 10 minims of tincture of opium after each cachet, provided that not more than 30 or 40 minims are given in twenty-four hours. Careful attention should be paid to the state of the alimentary canal, as constipation often appears to be a determining factor in bringing on pains.

Injections of morphia will, of course, give relief, but, on account of the danger of the patient acquiring a craving for this drug, they should only be resorted to in very exceptional cases.

The various crises must be treated by similar methods. In the **gastric crises** the difficulty of retaining drugs in the stomach sometimes makes it necessary to give relief by hypodermic injections or by rectal suppositories. In very severe cases division of the posterior roots of the nerves supplying the stomach (7th–11th dorsal) has been occasionally practised, but it is an operation of considerable severity, and uncertain in its results.

Treatment of bladder troubles.—When it is necessary to draw off the urine the utmost care must be taken to use an aseptic catheter. When cystitis has occurred, if the urine is acid, urotropine with or without acid sodium phosphate may be given; if alkaline, boric acid, ammonium benzoate, and acid sodium phosphate should be used. If necessary, the bladder must be washed out.

Treatment of ataxy.—To many patients the inability to walk straight is their most distressing symptom, and the methods introduced by Frenkel for correcting this difficulty by means of systematic exercises have proved to be of decided value.

Incoördination is the result of degeneration of nerve fibres from the muscles, joints, and ligaments whereby the sense of position of the limbs is greatly diminished, often so much so that when the eyes are closed the patient is unable to tell at what angle his arms or legs are flexed.

The object of Frenkel's exercises is to improve the sensibility of the remaining fibres so that their increased function will fill the gap left by those that are degenerated.

In an average person the functions of the special senses are seldom, if ever, educated to the highest possible pitch—a fact which is clearly shown by the delicate sensibility for touch and sound which may be acquired by the blind. In tabes, unless the degeneration is exceptionally extensive, there is a considerable margin left for the possibility of improving the muscle-sense, and by taking advantage of this possibility the incoördination can to a great extent be corrected.

To obtain success, the exercises must be performed slowly and accurately, great care being taken to avoid fatigue, which, as stated above, is so harmful. It is necessary to insist upon this point, because the efforts made to move the limbs slowly and accurately are a great strain on the mind as well as on the body.

Various exercises can be devised without much trouble and expense. The patient may be taught to regain command over his legs by lying on a couch and raising the foot from square to square of a wooden frame so that the big toe is brought to rest smoothly at each movement, and moving the foot backwards and forwards so that the heel is placed accurately on various marked spots. To this may be added exercises in walking accurately to patterns on the floor; while, later on, walking sideways and other more difficult feats may be attempted.

The hands may be trained by means of draught and halma boards, or some similar objects, by which accuracy and attention are brought to bear on the movements.

Schwab and Allison * have shown that in tabes the foot tends to assume a faulty position of eversion, and that by correcting this with suitable boots a mechanical advantage is obtained which further assists to diminish the incoördination.

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* *Journ. of Amer. Med. Assoc.*, Dec. 16, 1905.

SECTION VI.—DISEASES OF THE BRAIN, AND DISEASES OF GENERAL ORIGIN

CHAPTER XXXIV

MENINGITIS

INFLAMMATION of the membranes of the brain may occur as part of a general infective disorder, or it may arise from extension of some local disease of the skull. In the latter case the dura mater is especially liable to be affected (*pachymeningitis*), but in the former the primary seat of the inflammation is generally in the pia mater and arachnoid (*leptomeningitis*).

The micro-organisms which most frequently invade the meninges are those of tuberculosis, pneumonia, typhoid, septicæmia, cerebro-spinal fever and its sporadic form posterior basic meningitis. In many cases the primary infective organism becomes associated with streptococci and staphylococci during the course of the illness.

While the invasion of the meninges may be the predominating feature during life, an examination of the body after death very frequently reveals disease in other organs as well. Thus, tuberculous meningitis is practically always a part of a more generalized infection, as also are the cases due to the organisms of typhoid and, though perhaps less often, of pneumonia. The syphilitic forms have been already mentioned (Section V.).

I. TUBERCULOUS MENINGITIS

Etiology.—Tuberculous meningitis is most commonly found in children from 2 to 10 years of age, though its occurrence in adults is by no means rare.

The contributory causes are debility and the presence of tuberculosis of the lungs, glands, or some other part of the body.

Symptoms.—The early symptoms vary according to (1) the intensity of the general toxæmia, and (2) the prominence, or otherwise, of local symptoms.

As a rule, there are signs of *general disturbance* for some time before there is any reliable evidence of cerebral trouble, the child being irritable, languid, and generally out of sorts. An early sign of considerable importance is *vomiting* for which no adequate cause can be found, and with which is often associated *constipation*. The temperature is a little raised at night, and often runs an irregular course throughout the illness.

In other cases, distinct cerebral signs occur at an earlier date, and severe *headache*, with perhaps a *squint* and a general *convulsion*, may be among the symptoms by which attention is first attracted, though here again careful inquiry often elicits the fact that the child has not for some time previously been in its usual health.

The symptoms can best be remembered by considering what effects the *pathological* changes produce upon the various parts of the brain. The small, grey, semi-translucent tubercles are situated in the pia mater and are distributed chiefly over the base of the brain. They are very prone to occur along the course of the middle cerebral artery, and are generally most easily recognized between the opercula of the Sylvian fissure. They are usually much less numerous over the convexity of the brain, but in this respect their distribution is very variable in different cases.

Their growth is accompanied by a serous exudate of varying density. The chief effects produced by these processes are:

1. *Irritation* and *compression* of different structures of the brain.

2. *General rise* of *intracranial pressure* due to the exudation of fluid and the limitation of its circulation

pairment.

Optic nerve.—Optic neuritis
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Trigeminal nerve.—There are n
signs connected with the 5th nerv
will take the form of pain in th
distribution and of spasm or par
mastication.

Facial nerve.—Extension of th
trunk of this nerve gives rise to
the whole of one side of the fa
facial muscles may also occur if

Auditory nerve.—Deafness res
of the auditory nerve. It is not
symptom, for the impaired men
patient generally overshadows it.

Glosso-pharyngeal nerve.—Ther

upon other parts of the brain, and the variations of the respiratory rhythm will be again considered under that heading.

Spinal accessory nerve.—Irritation of the spinal portion of the spinal accessory is perhaps responsible to some extent for the retraction of the head, though probably this is also influenced by irritation of the cerebellum; but retraction of the head is often temporary, and in most cases is not a prominent feature of tuberculous meningitis, which is consistent with the fact that the exudation is frequently limited chiefly to the area of the circle of Willis, and does not extend sufficiently far down to implicate the cerebellum and the cervical nerve roots.

Hypoglossal nerve.—There are not usually any signs of disturbance of the function of the hypoglossal nerve.

The convexity of the brain.—When the inflammation extends on to the convexity of the brain there may be monoplegia or hemiplegia. Localized convulsions may also be a prominent symptom.

When we come to consider the effect of the **general increase of intracranial pressure,** we find a number of symptoms referable to compression and irritation of the cortex and meninges.

Pain.—The headache, perhaps slight in the early stages, may become so intense as to cause the intermittent shrieking known as the "hydrocephalic cry."

Vomiting.—Early in the disease the vomiting may be due to the general toxæmia, but in the later stages it is mainly the result of the rise of intracranial pressure, as in the case of cerebral tumours.

The *respiratory rhythm* is altered. Sometimes it is of the Cheyne-Stokes type, but more often a few respirations of equal intensity are succeeded by a short pause. The breathing is nearly always rapid.

The *pulse* for the most part is quickened and irregular, but in some stages of the illness it may be slow from irritation of the vagus, as already mentioned.

Convulsions are not uncommon. They arise from extension of the tubercles on to the cerebral cortex or from irritation induced by the general rise of pressure; localized twitchings also frequently occur.

The *abdomen* is *retracted*, and known as the "scaphoid abdomen."

There are pronounced *mental symptoms*; the patient is irritable, lies curled up on one side, avoids the light, and shows disinclination to answer questions or to be troubled in any way.

The *vaso-motor system* is deranged, and if the finger-nail be drawn over the skin it is rapidly followed by a flush that is known as the *tache cérébrale*.

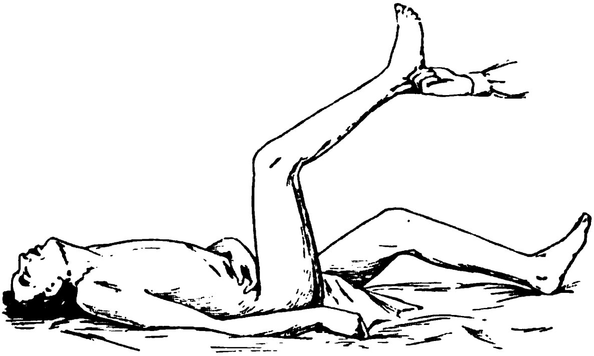

Fig. 85.—Method of obtaining Kernig's sign.
(After a photograph by Dr. W. Thyne.)

The *temperature* throughout the illness is somewhat irregular, and not generally very high; but towards the end there is often a considerable rise, which may continue for some time after death.

Kernig's sign.—The effects of cerebral irritation are seen in the increased "tone" (hypertonus) of muscles, and it is on this fact that Kernig's sign depends (Fig. 85). To obtain the sign the patient should be lying on the back, and, care being taken to keep one leg flat on the bed, the other should be flexed at the hip and knee. When the tone of the muscles is increased, the degree of extension which it is possible to obtain at the knee-joint while the thigh is flexed upon the abdomen is considerably less than normal. Kernig's sign is said to be present in over 80 per cent. of the cases of meningitis. It is, therefore, of considerable diagnostic significance, but it may, of course, be associated with other forms of cerebral irritation.

General course of the disease.—Attempts have been made to divide the disease into stages of irritation and compression, but the two conditions are necessarily concurrent, and it is very seldom that any sharp line can be drawn between them.

The duration of the symptoms is very variable. Death may occur within a week or ten days from the onset of acute symptoms, or it may be postponed for three weeks or more.

Prognosis.—Though cases have occasionally been recorded in which recovery has taken place from symptoms that seem to have been undoubtedly due to meningitis of tuberculous origin, such instances are so rare that for practical purposes the disease must be regarded as one which is always likely to cause death. Moreover, as already insisted upon, the meningeal inflammation is often only the more prominent part of a widely spread infection.

Diagnosis.—In children the chief difficulty in arriving at a diagnosis exists during the early stages of the disease. The child has perhaps been ailing for some time, but gets worse so gradually that it is difficult to assign the correct value to the vague symptoms of headache and gastric disturbance which so often precede those of a more definite character.

In other cases the difficulty is of the opposite kind, for the symptoms come on so acutely that for a short time it appears more likely that an acute specific fever is about to develop. Between these two extremes there is every gradation, so that it is frequently difficult to make a diagnosis with any certainty at an early stage of the disease.

Inflammatory troubles of the *middle ear*, the *mastoid*, and other parts of the skull may for a time be mistaken for meningitis, as also may the mental disturbance sometimes due to *intestinal worms*.

In adults the distinction between *typhoid fever* and tuberculous meningitis is often difficult. The main points to be relied upon can be tabulated as follows:

	Typhoid	Meningitis
Headache	Early symptom; tends to disappear later	Headache more intense; gets worse during the course of the illness. Hydrocephalic cry.
Optic neuritis ...	Not present	Frequent.
Ehrlich's reaction...	Generally obtained...	Exceptional, but may be present.
Widal's reaction ...	Usually obtained at the end of ten days	Not present.
Rash	Typhoid spots appear in crops from seventh to tenth day and onwards	Not present.
Squint	Not present	Very frequent.
Tubercle in other parts	Absent	Suggestive, though of course not necessarily diagnostic.
Temperature ...	Fairly regular course	More irregular.

Difficult cases can only be diagnosed by a careful review of all the symptoms in relation to one another, for nothing is more frequent than for exceptional and deceptive signs to appear in both these diseases.

The delirium of *lobar pneumonia*, especially of the apical form, is sometimes deceptive, and the knowledge that the pneumococcus may invade the meninges as well as the lungs makes the diagnosis still more uncertain in doubtful cases.

When the case appears to be one of meningitis, a careful search must be made for other causes before finally concluding that it is tuberculous. The ears must be examined for signs of suppuration, and any possibility of a cerebral abscess must be carefully considered. It is most important not to overlook any of these other causes of meningitis, since they may often be relieved by surgical treatment when the operation is undertaken in time.

Lumbar puncture may be an aid to diagnosis. In tuberculous meningitis the fluid is generally clear or only slightly flaky. Well-marked lymphocytosis is usually present, and in a considerable number of cases tubercle bacilli can be detected by staining after centrifugalization.

Treatment.—From the foregoing paragraphs it may be gathered that no drugs have any specific influence on the disease. Urotropine may be given for its supposed antiseptic action on the cerebro-spinal fluid. Mercury given by inunction has been recommended, but it is very doubtful if it is of any value. Potassium iodide is useless in tuberculous meningitis, but should be given if there is any suspicion as to the affection being syphilitic. Relief of pressure is sometimes obtained by lumbar puncture, and in this way a temporary amelioration of the symptoms may be secured; but the adhesions at the base of the brain usually prevent the drainage being sufficiently free to give any permanent relief.

The following general measures should be adopted: The patient should be kept in a dark room, the head shaved, an ice-bag applied, and plenty of nourishment given. Phenacetin, antipyrin, salicylates, and in severe cases opium, may be given for the relief of headache. The bowels should be kept open.

Signs of heart failure will indicate the necessity for digitalis and other suitable stimulants.

II. EPIDEMIC CEREBRO-SPINAL MENINGITIS (SPOTTED FEVER)

Etiology.—Epidemic cerebro-spinal meningitis (cerebro-spinal fever or spotted fever) is most prevalent among children and young adults.

The micro-organism, the *Diplococcus intracellularis* of Weichselbaum, commonly spoken of as the " meningococcus," is a kidney-shaped gram-negative coccus, and is now considered to be identical with that found in cases of posterior basic meningitis, described later in this chapter.

The exact conditions which cause the disease to assume an epidemic character are not known, but atmospheric changes, overcrowding, and chronic catarrhal states are probably factors of importance in modifying the virulence of the micro-organism. Epidemics are most frequent in the winter and spring months.

The disease is infectious, and is undoubtedly spread to a great extent by " carriers," who harbour the micro-organism in the throat, and so give the disorder to others without necessarily suffering from it themselves. The organism does not seem to possess great vitality outside the body, but, as it is often to be found in discharges from the nose and also in the expectoration of patients, care must always be taken to prevent the disease from spreading by these means.

The micro-organism is not very easy to cultivate, and grows best on serum agar. During life it can be obtained from the spinal fluid by lumbar puncture, but in many instances the results of bacteriological examination of the cerebro-spinal fluid are negative, especially if the cultivation is not made quickly after the fluid is withdrawn.

Pathology.—The meninges, brain, cord, and peripheral nerves (both cranial and spinal) are all apt to be affected. The pia and arachnoid membranes are the chief seat of the infection, and the exudation, which may be serous or purulent, is generally most intense at the base of the brain, although it often extends some distance over the cerebral hemispheres and the lobes of the cerebellum. In the most acute cases, nothing more than intense injection of the pia mater may be seen with the naked eye, but microscopical examination generally shows that a considerable amount of exudation has already take place.

In the chronic cases the membranes are often very much thickened, and have formed adhesions which may interrupt the circulation of the cerebro-spinal fluid and lead to hydrocephalus.

Similar changes are found in the pia mater and the arachnoid of the cord, more especially on the posterior surface.

Patches of softening and hæmorrhages are often present in both the cerebral and spinal substances.

The inflammation is also very apt to extend along the cranial and spinal nerves, which become swollen and softened in consequence. It is stated that the 2nd, 5th, 7th, and 8th are the most liable to suffer, and that inflammatory changes of an advanced character can generally be found in the Gasserian ganglia.

Symptoms.—The meningeal symptoms are often the first to which attention is directed, though in many cases the history shows the previous presence of some naso-pharyngeal catarrh, and in other instances symptoms of general septicæmia precede those of meningeal infection. The onset of meningitis is usually very acute, and of the early symptoms the most prominent are *vomiting* and *pain*, the latter being either localized to the head or of a generalized character.

The presence of meningitis of the brain and cord is soon shown by *palsies of the cranial nerves*, which cause strabismus, ptosis, unequal and fixed pupils, facial paralysis, and deafness; by *rigidity of the neck and trunk muscles*, with retraction of the head and opisthotonos; by *optic neuritis, convulsions, hydrocephalic cry, Cheyne-Stokes respiration, delirium*, and *coma;* and sometimes by *hemiplegia* and *paraplegia*. The mechanism by which these symptoms are produced, i.e. by irritation and compression, has already been discussed in the section on Tuberculous Meningitis, to which the reader is referred.

Inflammatory conditions of the *eyes*, *nose*, *ears*, and *lungs* are often present. The eye symptoms are very important, and include conjunctivitis, iritis, corneitis, and sometimes inflammation of all the structures of the eyeball, which may result in permanent blindness. Apart from these changes, a temporary loss of vision may occur. Disease of the ears is a complication frequently met with; the internal ear may be damaged by the extension of the inflammation along the auditory nerve, to which reference has already been made; and there is often acute inflammation of the middle ear, in the discharge from which the specific micro-organisms may sometimes be found.

Not infrequently there is a discharge from the nose, from which the diplococci may also be isolated. The bony sinuses of the skull, too, are frequently infected.

The organism may reach the lungs, and produce broncho-pneumonia.

Small hæmorrhages into the *skin*, forming petechial spots, are frequently present; hence the name "spotted fever." The presence of herpes has also been frequently observed.

The *pulse* and *temperature* are very variable. Swelling of the *joints* sometimes occurs.

Clinical course.—In the *fulminating* cases, death may take place within twenty-four hours. The ordinary acute cases generally exhibit all the main symptoms that have been detailed, and when they are about to terminate fatally the coma deepens and all power over the sphincters is lost, as in other forms of meningitis.

Sometimes the disease runs a more *chronic* course, and is accompanied by great emaciation and weakness, and—if recovery takes place—by impairment of the mental faculties or hydrocephalus.

Cases in which there are intermission and remission of symptoms are sometimes observed.

Diagnosis.—Differentiation has to be made between this disease and other forms of meningitis, such as those due to the tubercle bacillus and the pneumococcus.

To decide the diagnosis, lumbar puncture should be performed, and the fluid thus obtained should be examined for the diplococci. The presence of these makes the diagnosis conclusive; but too much importance must not be attached to a negative result, for the organism is cultivated with difficulty, and may be absent during the remissions and also in the later stages.

Prognosis.—The death-rate of those who develop meningitis is very high, but varies in different epidemics according, presumably, to the virulence of the organism. In individual cases the prognosis must be based upon the intensity of the general infection and upon the signs denoting the degree of intracranial pressure.

Treatment.—All suspected cases should be isolated and search instituted for "carriers" by making bacteriological examinations from the throats of contacts. Antimeningococcus serum, as first recommended by Flexner, has proved of great benefit, and the mortality among those to whom the serum has been given has been greatly reduced as compared with that of patients not so treated. A lumbar puncture should first be performed, after which the serum, adjusted in amount to the quantity of cerebro-spinal fluid withdrawn, should be slowly introduced, preferably by the gravity method.

An attempt to relieve the intracranial pressure may be made by performing lumbar puncture at short intervals, and in some cases this procedure appears to assist materially in tiding the patient over the critical period of the disease.

III. Posterior Basic Meningitis

Etiology.—In this form of meningitis the inflammation is mainly confined to the posterior part of the base of the brain. The micro-organism, first noted in connection with this disease by Still, has proved to be identical with the meningococcus of epidemic cerebrospinal meningitis, of which posterior basic meningitis must be considered to be a sporadic form.

The organism probably reaches the membranes of the brain by way of the Eustachian tube and middle ear, and any debilitating process, such as rickets, injury, or catarrh, renders the child more liable to infection.

Children of both sexes are attacked, and the majority of cases occur during infancy.

Pathology.—The exudate is found over the posterior part of the base of the brain, and, if it has existed long enough, there may be occlusion of foramina in connection with the 4th ventricle and interruption of the flow of cerebro-spinal fluid; the latter then collects in the lateral ventricles and produces hydrocephalus. Sometimes the inflammation extends some way down the cord or on to the vertex of the brain.

Symptoms.—The *onset* is usually *acute*, but the symptoms may be indefinite for a time. *Vomiting* occurs early, and is generally accompanied or quickly followed by *retraction of the head*, which is one of the most prominent features of the disease (Plate 15).

More widely spread tonic spasms cause general rigidity, or even opisthotonos; convulsions may also occur. The rise of intracranial pressure is shown by the tense fontanelles, and it is probably the cause of the temporary blindness which is often present, since optic neuritis is seldom met with. The condition of the pupils varies; in some cases they are contracted, in others dilated. Owing to retraction of the upper lids, the eyes often appear unduly prominent; nystagmus may sometimes be observed. Strabismus often occurs, though not with such frequency as in tuberculous meningitis.

Alterations resembling those of tuberculous meningitis may take place in the pulse and respiration.

The deep reflexes vary in their reactions, and are sometimes increased, sometimes absent.

In the later stages the mental powers may be impaired by hydrocephalus.

Diagnosis.—At the onset it is often difficult or even impossible for a time to decide whether meningitis or some other acute febrile condition, e.g. pneumonia, is present. After a few days the reference of symptoms to the head, together with the absence of signs of a generalized infection, makes the locality of the disease

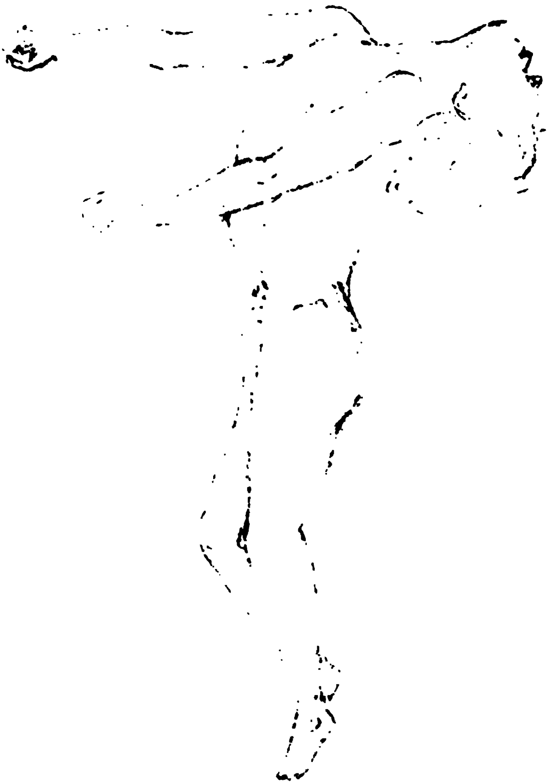

PLATE 15.—Case of posterior basic meningitis showing extreme retraction of the head.

(*From a sketch by Dr. Penrose Watkin Williams.*)

more apparent, and it is then necessary to exclude the presence of *sinus thrombosis, otitis media,* and *mastoid disease* before arriving at the diagnosis of meningitis. When the diagnosis of meningitis has been made, it is still necessary to attempt to decide the nature of the infection, and the chief difficulty lies in differentiating the posterior basic from the tuberculous form.

Reference to the list of symptoms shows that in posterior basic meningitis the rigidity and retraction of the head are the most prominent symptoms, while optic neuritis, palsies of cranial nerves, and retraction of the abdomen are less likely to be present. The detection of tubercles in the choroid will, of course, decide the question, as also may a bacteriological examination of the cerebro-spinal fluid. A blood-count may also be of value, for in posterior basic meningitis leucocytosis is usually greater than that which occurs in tuberculous meningitis.

Prognosis.—The prognosis of posterior basic meningitis is not altogether unfavourable. A considerable number of patients get well, though often with mental impairment, the result chiefly of the attendant hydrocephalus. The duration of the illness varies, but it often lasts several weeks, and thus may be longer than in the tuberculous form.

Treatment.—Mercury may be given on account of its general tendency to influence inflammatory processes, and it is well borne by children. Iodides have also been recommended. Operations with a view to draining off the excess of fluid and relieving the pressure have not met with any constant success, but cases have been recorded in which repeated lumbar punctures have been followed by beneficial results.

IV. SEROUS MENINGITIS

In this variety, which is also known as " wet brain," the exudate is serous in character and often more œdematous than inflammatory. The condition has been more particularly described in association with chronic alcoholism, but it may be found in other circumstances, and more recently probable cases have occasionally appeared to occur in association with attacks of lethargic encephalitis (*see* p. 346).

The **symptoms** may resemble those of other forms of meningitis, but are usually less intense. The general signs of increased intracranial pressure are shown by headache, optic neuritis, and vomiting, with which may be associated various signs of cranial nerve disturbance. When, as sometimes happens, the temperature is subnormal the condition may closely resemble cerebral tumour.

With rest the symptoms may subside, and relief is also sometimes obtained by lumbar puncture. If no improvement takes place, or if the vision is in danger of permanent deterioration, the question of performing an operation for cerebral decompression should be considered.

V. SUPPURATIVE MENINGITIS

Etiology.—The invasion of the meninges by pyogenic organisms may take place from extension of local suppuration, or it may be a part of the terminal stage of a general infection. The local conditions which most frequently give rise to meningitis are suppurative otitis media, mastoid disease, and cerebral abscess, all of which are frequently closely associated with one another.

The **symptoms** of suppurative meningitis do not materially differ from those described under the heading of Tuberculous Meningitis, since they also depend upon the irritation and pressure of exudation. The general tendency, however, is for the suppurative forms to spread over the convexity of the brain rather than to remain confined to the base; hence the functions of the cranial nerves are apt to be less extensively deranged than in the tuberculous form.

Suppurative meningitis is accompanied by severe general symptoms, and when diffused always causes death.

The **treatment** consists in relieving the symptoms on the lines already mentioned. When the meningitis is once established the opportunity for surgical interference has passed, and it is, therefore, most important to deal promptly with any local suppurative process about the skull before any extension to the membranes has taken place.

VI. Pachymeningitis Externa

In some cases where there is direct extension from the bones of the skull the suppuration is for a time, at any rate, confined to the outer surface of the dura (pachymeningitis externa), and if a collection of pus be localized, incision and drainage may be attended with success. Left to itself, the natural tendency is for the pus to infect the inner membranes.

VII. Injuries to the Meninges

Among the common causes of injuries to the meninges are damage to the skull during childbirth and, in later life, blows on the head. One of the most serious injuries is a blow on the head causing hæmorrhage from the middle meningeal artery or one of its branches. The immediate effects of a blow of this kind may not appear to be serious, but after a brief interval of time the results of the hæmorrhage make themselves felt. The patient becomes dazed and soon loses consciousness; signs of progressive hemiplegia, with or without convulsions, develop; the loss of consciousness deepens, and the patient rapidly becomes comatose and dies unless the skull can be opened and the bleeding quickly arrested.

Experience of war injuries has shown that damage to the membranes and to the cortex beneath may be produced by high velocity missiles merely grazing the bones. Wilfred Trotter * has investigated this condition and concludes that " the resilience of the skull is such that whenever a missile travelling at high velocity touches the bone, the latter is driven in upon the brain with force enough to inflict a considerable bruise," and that in such circumstances a contusion of the brain is likely to remain longer than it would in other organs, owing to the unyielding character of the cranium, which prevents the compensatory swelling by which the circulation is maintained through other bruised organs. The principal symptoms in such a case are headaches, made worse by exertion, especially stooping, and giddiness, which symptoms, Trotter finds, can be greatly relieved or cured by free removal of bone and incision of the dura mater at the seat of injury, the opening being closed again some months later. On operation the arachnoid is found to be opaque and distended by a local accumulation of fluid beneath it, and the surface of the brain is discoloured from old extravasated blood.

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* *Brain*, vol. xlii., pt. iv., 1919.

CHAPTER XXXV

CEREBRAL PALSIES OF CHILDREN; AMAUROTIC FAMILY IDIOCY; HYDROCEPHALUS

CEREBRAL PALSIES OF CHILDREN

THE paralyses of cerebral origin which occur in childhood may be grouped, according to their causation, as follows:

- (*a*) *Before birth:* e.g.
  - (1) Primary arrest of development.
  - (2) Changes depending on maternal syphilis or some other toxic condition.
- (*b*) *During birth:* e.g.
  - (1) Hæmorrhages into the meninges.
  - (2) Direct injury of cerebral cortex.
- (*c*) *After birth:* e.g.
  - (1) Acute encephalitis.
  - (2) Thrombosis.
  - (3) Meningeal hæmorrhage.
  - (4) Meningitis.
  - (5) Cerebral tumours.
  - (6) Nerve degeneration.

Pathology.—As might be expected from the variety of the causal lesions, the brains differ very markedly from one another in their appearance.

In some, an abortive development of the motor areas is the main feature—a condition known as cerebral agenesis. In others, the brain is small, and exhibits a generalized sclerosis; while in others again, cystic conditions, known as porencephaly, are found.

Thrombosis of vessels and softening of brain tissue occur from encephalitis, and may give rise later on to a scar, over which the membranes may be thickened and adherent.

The paralysis usually takes the form of a diplegia or of a hemiplegia.

CEREBRAL DIPLEGIA

Cerebral diplegia is characterized by spasticity of both sides of the body, and by a variable degree of weakness. Arrested development of the motor areas during intra-uterine life appears to be the most common cause. The nerve elements which should make up the motor areas of the brain either fail to develop, or degenerate during the period of their development, poisoned by syphilis or some other toxin. In some cases they appear to atrophy from sheer want of vitality, and so form a connecting link with those in which the degeneration occurs in later childhood—e.g. in Friedreich's disease and some of the myopathies—a connection which is further emphasized by the occasional tendency of cerebral diplegia to appear in different members of the same family.

All gradations exist between complete absence of the motor areas and slight impairment of their functions, with corresponding clinical variations. When the damage to the brain occurs during the birth of the child, it is most probably due to a hæmorrhage into the meninges whereby the motor areas of the legs are more or less symmetrically injured, the lesion sometimes extending as far over the hemispheres as the areas corresponding to the movements of the upper extremities.

After birth the injuries are far more likely to be unilateral, and to cause hemiplegia rather than diplegia.

It very frequently happens that the exact period at which the damage occurred cannot be ascertained with any degree of certainty from the history of the case.

Symptoms.—The main symptoms of cerebral diplegia are *rigidity, weakness, involuntary movements,* and *mental infirmity,* but their variability in degree is very great, so that cases differ very considerably from one another.

In a typical case of cerebral diplegia it is noticed that the child's limbs are stiff, and that the movements are not so free as they should be.

On examination, there is evidence that the child has the signs of degeneration of the motor tracts, as shown by weakness, rigidity, and increased knee-jerks. The behaviour of the plantar reflex is not of much assistance in young infants, since in them it is normally of the extensor type, but in older children it possesses the usual significance.

As a rule, the rigidity is rather out of proportion to the weakness, and, on the patient attempting to walk, the well-known "scissors-legged" attitude is assumed, owing to predominant spasm of the adductors of the thigh (Fig. 86). The arms share in the rigidity, though oftentimes to a less degree. The face may also be affected, and occasionally there is coincident atrophy of the optic nerves.

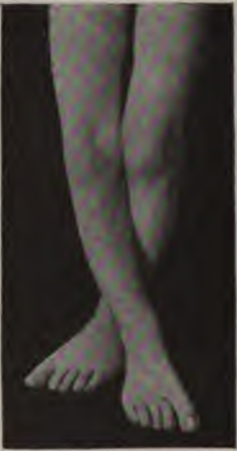

Fig. 86.—"Scissors-legged" progression in a case of cerebral diplegia.

The weakness, always present in some degree, is often rather masked by the rigidity which makes voluntary movements far more difficult than they would otherwise be.

Involuntary movements occur in a considerable number of cases, and often attack the hand and arm (less commonly the leg and face), taking the form known as athetosis, the chief characters of which are slow, more or less rhythmical "squirming," involuntary movements of the fingers and thumbs, in which hyperextension is a prominent feature. Sometimes the whole arm takes part in the movements, which may be so violent and uncontrollable as to necessitate strapping the limb to the side. Other movements may take the form of tremors, or may be of a more irregular choreiform nature (Plate 16).

All degrees of mental impairment are met with, from " backwardness " to complete idiocy. Articulation also is often impaired.

Epileptic fits are frequently associated with cerebral diplegia.

The less severe cases of diplegia, in which the symptoms are confined to the legs, and consist of some rigidity combined with weakness of only moderate degree, are often classed together in a group under the heading of Little's disease, after the name of the surgeon who first described them. An "atonic" form, in which myatonia is a prominent symptom, has also been described.

Diagnosis.—When the symptoms are pronounced, there is but little difficulty in recognizing their nature.

The limbs of children suffering from *severe rickets* or *scurvy rickets* sometimes appear to be paralysed, but the absence of rigidity, with the enlargement of the ends of the long bones, and other signs of rickets, shows that the weakness is not of nervous origin.

In older children, with symptoms of a moderate degree, disseminated sclerosis and Friedreich's disease may require consideration. *Disseminated sclerosis* is very rare in childhood, and possibly some of the cases recorded have been, in reality, cerebral diplegias.

Variability of symptoms and nystagmus may help to distinguish difficult cases, and, of course, the history, when obtainable, is a most reliable aid.

It is important to differentiate the two diseases carefully, since the outlook for those suffering from disseminated sclerosis is not good, whereas the slighter forms of cerebral diplegia often tend to improve.

Friedreich's disease comes on later in life, and the ataxy, loss of deep reflexes, nystagmus, and other

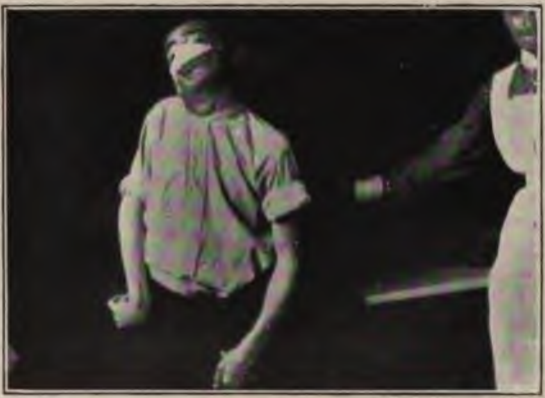

PLATE 16.—Instantaneous photograph showing bilateral athetosis in a case of cerebral diplegia
(*By courtesy of Dr. George Ogilvie.*)

characteristic symptoms will generally enable it to be distinguished.

Prognosis and treatment.—Extraordinary improvements can sometimes be effected in the less severe cases by patiently persevering with mental and physical education; for the latter carefully regulated gymnastics should be prescribed. Pronounced mental impairment and frequent fits are of evil significance, and the onset of athetosis usually indicates that the chance of further improvement in the use of the limbs is but slight.

In cases where the difficulty in using the limbs is due to excessive rigidity rather than loss of power, improvement has sometimes been obtained by Foster's operation of dividing the posterior nerve roots. By this procedure the rigidity may be materially diminished, but care must be taken to divide only a certain number of nerve roots to each limb, for were all divided the part would be rendered useless through the total anæsthesia and loss of muscle tone that would naturally result.

In other instances the power of movement may be improved by stretching or dividing the spastic muscles.

All cases for operation should be carefully considered from the mental as well as the physical aspect of the patient, for improvement ultimately depends largely upon the success with which postoperative treatment by systematic exercises and re-education of movements can be carried out.

Infantile Hemiplegia

The hemiplegias of childhood are, for the most part, due to injuries received at the time of birth or to postnatal inflammatory and vascular lesions.

During prolonged and difficult labours, hæmorrhage or some other injury may occur by which the functions of the cortical motor areas are impaired.

After birth, certain blood states, especially those brought about by specific fevers, are conducive to thrombosis in the cerebral vessels, or the direct invasion of the brain tissue by micro-organisms may give rise to an acute encephalitis. The most important of the latter are the forms known as *acute polioencephalitis*, in which the toxin appears to be identical with that of acute poliomyelitis (*see* p. 189), and encephalitis lethargica (*see* p. 341).

Symptoms.—Of the cases due to polioencephalitis, most occur in children between the ages of 3 and 6, and the onset is generally marked by fever, malaise, and convulsions, with more or less disturbance of consciousness.

Between the convulsions that attend the onset of the disease, or more frequently after the lapse of one to several days, unilateral weakness is observed, generally most pronounced in the upper extremity; and in some cases the motor nuclei of cranial nerves are affected like those of the cord in anterior poliomyelitis.

Sometimes the power gradually returns to the affected parts, but in the majority of cases a considerable amount of paralysis persists. The later symptoms are, in the main, those already described in the diplegias, viz. fits, mental deterioration, and perhaps athetosis.

When the lesion is in the cortex of the left hemisphere there is the possibility of aphasia, but the right side will probably take up the function of speech if the subject is a young child.

Encephalitis lethargica is less common in childhood, but the lasting effects of any local damage to the brain from this cause will be similar to those of polioencephalitis described above.

Diagnosis.—The convulsions and distribution of the paralysis indicate a cerebral, as opposed to a spinal, lesion, while fever, and drowsiness or complete loss of sensibility, are the usual accompaniments of cases of acute polioencephalitis. The presence of a valvular lesion of the heart will suggest the possibility of embolism.

When a long-standing case is seen for the first time, its cerebral nature will be evident from the spasticity, increased reflexes, and absence of electrical changes, in contrast to the flaccidity, the loss of reflexes, and the reaction of degeneration found in the paralysis following acute anterior poliomyelitis of the cord. The atrophy is much more evident in cases following the latter lesion, although the limbs of the infantile hemiplegic also look wasted owing to arrest of their development, and club-foot is a common occurrence in both diseases.

Treatment.—During the febrile period of cases of acute encephalitis the child should be kept quiet in bed and the room darkened. A mixture containing bromides and a purge of calomel or grey powder should be given, and the diet should be as light as possible. When the signs of cortical irritation have quite disappeared, the most careful and persistent attention must be given to the paralysed limbs. Passive and, where possible, active movements must be practised regularly, and every possible effort should be made to prevent the onset of contractures.

The ultimate results in many instances are surprisingly good, but no accurate prognosis can be given for any individual case, since the onset of fits, athetosis, or permanent mental disability may mar the most promising outlook.

When fits occur they must be treated with bromide, as in ordinary epilepsy.

The possibilities of treatment by surgical methods are similar to those discussed in the preceding section dealing with diplegia.

Cerebellar Palsies of Childhood

The **causes** of the cerebellar lesions of childhood are very similar to those producing the cerebral palsies, and may, in like fashion, be divided into those occurring—

(*a*) Before birth.
(*b*) During birth.
(*c*) After birth.

The main symptom in cases of cerebellar disease in children is **ataxia**, and F. E. Batten * has shown that cases of ataxia in childhood in relation to cerebellar disease may, from a clinical point of view, be classified as follows :

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* *Brain*, 1905, Pts. cxi., cxii.

(1) *Congenital cerebellar ataxia ;* in which the ataxia has been noticed from earliest life.
(2) *Acute cerebellar ataxia ;* due to encephalitis of the cerebellum, in which the ataxia has developed during the course of or after some acute illness in a child who was formerly healthy.
(3) *Progressive cerebellar ataxia ;* in which the ataxia has gradually developed in a child who has previously been healthy and of normal development.

The value of this classification is enhanced by F. E. Batten's observations on the **prognosis,** which led him to conclude that the cases which come under the heading of congenital ataxia, and those also which are due to polioencephalitis, show a tendency to improve gradually, while in those belonging to the group of progressive ataxia the outlook is bad.

AMAUROTIC FAMILY IDIOCY

(WAREN TAY'S DISEASE; SACHS' DISEASE)

This disease, which Sachs has termed amaurotic family idiocy, is characterized by progressive degeneration of nerve cells throughout the whole of the nervous system.

Etiology.—There is a great tendency for the disease to appear in several members of a family, and hitherto all the recorded cases about the nature of which there is no doubt have been children of Jewish parents. The children, apparently healthy when first born, develop symptoms within the first few months.

Different views are held concerning the probability of a congenital or an acquired origin of the disease : the family characteristics certainly point to a congenital origin, and the idea that abnormalities of the mother's milk may be a cause appears to have been disproved by

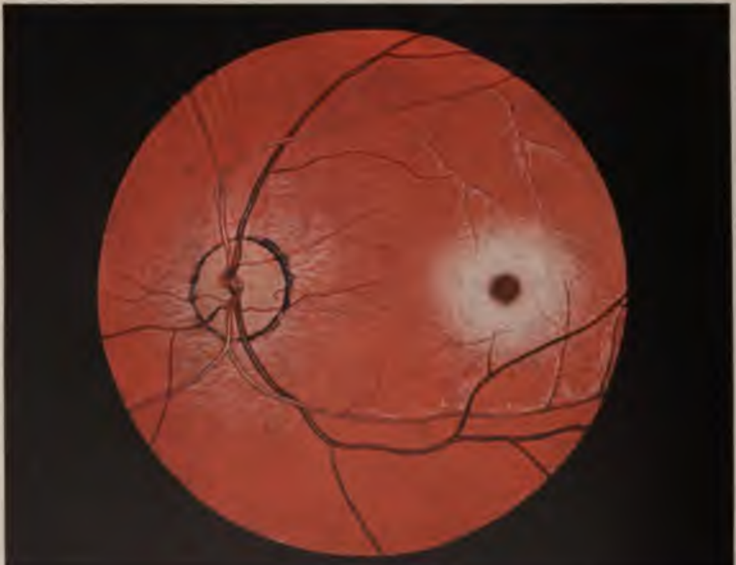

PLATE 17.—The fundus oculi, showing the characteristic appearance in amaurotic family idiocy.

(By courtesy of Mr. Ilbert Hancock.)

Sachs, who caused several of his patients to be reared by wet-nurses. Gordon Holmes and Poynton, after careful consideration of cases which they have examined, think that the disease is due neither to arrested development nor to bacterial toxins, but depends upon "some inherent biochemical property of the protoplasm of the cells" which brings about the degenerative processes.

Pathology.—There is a widespread degeneration of the cellular elements of the brain cortex, with various degrees of sclerosis. Changes have also been noted in the basal ganglia and grey matter of the cord. Mott * concludes that the essential feature is failure in the elaboration of the nucleo-proteins of the neurones, as evidenced histologically by the disappearance of the Nissl granules.

Symptoms.—The symptoms usually appear in the first few months of life—mostly between the third and sixth. The child becomes *weak* and *wasted,* and there is soon an obvious *paralysis,* which affects most of the body, and which may at first be of flaccid, and later on of spastic type. At the same time the power of *vision* fails and the *mental faculties* rapidly deteriorate.

The wasting and weakness progress, and death usually takes place in a few months.

The condition of the *reflexes* varies with the spastic and flaccid nature of the paralysis.

The *ocular* symptoms, first described by Waren Tay, are very characteristic of the disease, for, in addition to optic atrophy, there is in the region of the macula lutea a whitish patch, in the middle of which is a round spot of cherry-red colour. The appearance of this is similar to that occurring after embolism of the central artery of the retina (Plate 17).

Prognosis and treatment.—Up to the present, no methods of retarding the course of the degeneration have been discovered.

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* *Arch. of Neurology*, vol. iii., 1907.

HYDROCEPHALUS

Hydrocephalus is characterized by the accumulation of serous exudation within the skull.

Internal hydrocephalus is the form usually met with, and may be the result of many different underlying pathological conditions.

External hydrocephalus is the term applied to the collection of fluid between the dura mater and the brain, but the symptoms to which it gives rise are usually masked by the more important changes on which it depends.

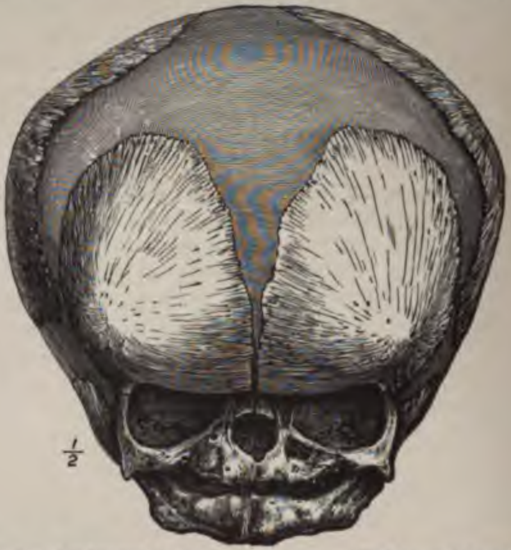

Fig. 87.—Hydrocephalic skull. (*Museum, Middlesex Hospital; Sir John Bland-Sutton.*)

Acute hydrocephalus occurs in association with various forms of meningitis.

Chronic hydrocephalus may be due to some congenital condition, or it may arise after birth as the result of meningeal inflammation or tumours. In many cases the chief factor seems to be obliteration of the foramen of Magendie and other passages through which the fluid normally circulates, added to which there may be an excessive secretion of the fluid.

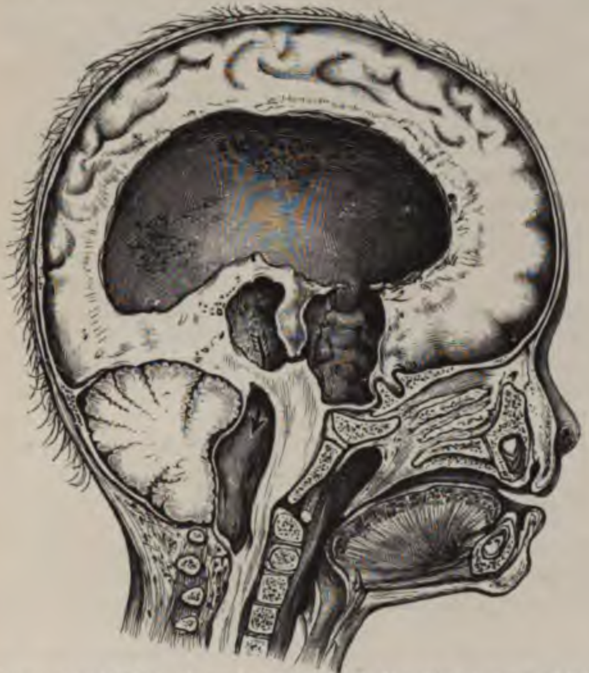

Fig. 88.—Sagittal section of a hydrocephalic skull from a child, with the brain *in situ*. The head of the arrow is in the 4th ventricle, and its feathers are in the 3rd. The infundibulum is widely dilated. (*Museum, Middlesex Hospital; Sir John Bland-Sutton.*)

The distension of the ventricles usually causes alterations in the brain, leading to mental impairment, but in some instances the intellect has remained good. Various degrees of diplegia may also be present.

further investigation showed that there was no ground for this belief. Careful consideration was next given to the question whether the symptoms were another manifestation of the group known as the Heine-Medin disease, of which acute poliomyelitis is the best-known variety, but sufficient differences were found in the age-incidence, seasonal prevalence, course, duration and termination, as well as in the localization of the paralyses, to make it clear that the condition is not identical with that of acute poliomyelitis.*

The exact nature of the infection has not yet been determined, but it appears probable that the pathogenic organisms, like those of cerebro-spinal fever and acute poliomyelitis, are frequently present in persons who show no clinical signs of disease until their resistance is lowered or the virulence of the organism is increased.

McIntosh and Turnbull† and others have succeeded in producing the disease in monkeys by inoculation of brain emulsion from a fatal case in man.

Symptoms.—The length of the incubation period is uncertain and appears to be variable. The prodromal period, during which such symptoms as lethargy, headache, giddiness and diplopia, with vomiting, diarrhœa, and general sensations of fatigue, may occur before the more acute stage of the disease is reached, commonly ranges from one to seven days but may last as long as three weeks (MacNalty). The acute stage is characterized by the onset of stupor, delirium, and other symptoms which differ according to the intensity of the infection and the locality of the nervous lesions accompanying it.

It will thus be seen that the symptoms differ very considerably in different cases. On the one hand there are the abortive cases, in which there may be nothing more than a vague illness characterized by apathy, sleepiness, and slight pyrexia, associated with some temporary paresis of ocular muscles; while on the other hand there are the severe cases accompanied by extreme prostration, coma, paresis of various cranial nerves, and other signs of severe damage to the nervous system.

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* *Brit. Journ. Exper. Path.*, April, 1920.

† *Ibid.*

The *onset* may be gradual or rapid. In the early stages of a typical case, conjunctival and pharyngeal catarrh, headache, giddiness, muscular pains, gastric and intestinal disturbance (especially constipation), together with some degree of pyrexia, are all commonly found, but the most characteristic symptom is that of lethargy, which tends to increase in depth. In some instances it does not amount to more than an overwhelming desire to fall asleep at inconvenient times and places, while in others it deepens into a state of coma from which the patient cannot be roused.

As the illness progresses the general symptoms of bodily weakness become more pronounced and the features assume a striking wooden, mask-like expression, somewhat similar to that seen in the later stages of paralysis agitans. In the more severe cases the patient becomes delirious, the excreta are passed unconsciously, the breathing becomes stertorous and altered in rhythm, the circulation shows signs of failure, and finally, if the disease causes a fatal termination, death takes place usually towards the end of the third week of the illness.

Side by side with these general symptoms there are various paralyses depending for their characteristics on the locality of the brain attacked.

Of the cranial nerves, paresis of the third, as shown by ptosis and limitation of movements of the eyeball, is most commonly found, but any of the other nerves may also be affected, and such symptoms as deafness, paresis of the soft palate, and weakness of the tongue are all met with, as also are signs of disturbance of the vagus. Optic neuritis, due apparently to rise of intracranial pressure from exudation, has also been observed. When the cerebral cortex is affected paralyses of monoplegic, hemiplegic, and diplegic distribution, and disturbances of special senses such as hemianopia, are among the symptoms likely to be found, while disease of the corpus striatum will be associated with tremors, rigidity, and involuntary movements. In some cases the occurrence of rhythmical muscular contractions (so-called "myoclonus") have been observed. Lesions of the medullary region are accompanied by failure of the heart and circulation.

Prognosis.—The outlook in cases of any degree of severity is one of danger. Improvements are often followed by relapses, so that the patient, even when seemingly well on the way to recovery, cannot be considered as entirely out of danger.

Convalescence is slow, and influenced largely in its course by the nature of the nervous lesions, some of which may be of a more or less permanent and crippling nature. Cranial nerve palsies may, for instance, persist, or there may be some degree of hemiplegia remaining, but among the most striking results of a more lasting nature are the involuntary movements of limbs and the rigidity and tremors of paralysis agitans type, both varieties apparently being due to disease in the region of the corpus striatum. The involuntary movements which may occur in the face, arm, and leg resemble in their general characters the athetosis met with after cases of hemiplegia. (*See* p. 399.) The movements are usually most prominent in the arm, and may either be confined to the hand or, as frequently happens, the whole limb may be thrown about irregularly or move with a kind of jerky regularity.

The cases resembling the symptoms met with in advanced stages of paralysis agitans are very striking, and are accompanied by the weakness, rigidity, and tremors so characteristic of that disease. The general attitude of the patient is well shown in Fig. 89, which should be compared with the illustration of the attitude in a case of paralysis agitans (Fig. 111, p. 455).

The general tendency in these cases is for some improvement slowly to take place, but in many, more especially in older people, the patient is to a large extent permanently crippled.

Morbid anatomy.—The appearances of the brain after death have been found to vary a good deal in different cases. Farquhar Buzzard and Greenfield* have investigated numerous fatal cases and found the most common conditions present during the early stages to be (1) vascular congestion, (2) evidence of toxic degeneration of nerve cells, (3) proliferation of the mesoblastic cells of the vessel walls and infiltration of the nervous tissue with these cells, (4) small-celled infiltration of the Virchow-Robin spaces, and (5) glial proliferation.

Venous thrombosis and hæmorrhages were also found by Buzzard and Greenfield in a certain number of cases; in some instances the hæmorrhages occurred as rings round congested vessels, and in others they were the results of infarction.

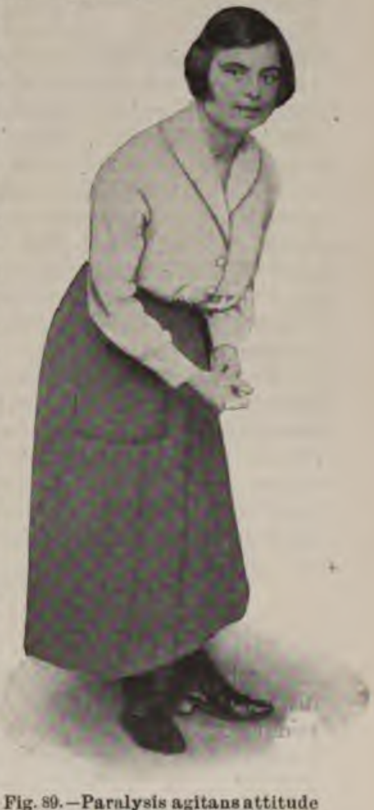

Fig. 89.—Paralysis agitans attitude after encephalitis lethargica.

Diagnosis.—In the early stages, when vague symptoms are accompanied by pyrexia, the illness may be indistinguishable from *influenza* unless some characteristic symptoms such as ocular palsies or excessive drowsiness arise.

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* *Brain*, vol. xlii., pt. iv., 1919.

An inflammatory lesion in the course of the motor fibres may give rise to hemiplegia, while disturbances of the sensory fibres may be accompanied by hemianæsthesia or hemianopia, all of which are apt to be ascribed to results of primary vascular changes, such as *thrombosis* or *hæmorrhage*, unless the possibility of encephalitis is kept in mind. *Meningitis* and *cerebral tumour* may also be simulated by the severe headache and other signs of increased intracranial pressure, including optic neuritis, that sometimes occur from the exudation of serous or bloodstained fluid, and if they persist after the acute signs of general infection have subsided, the clinical signs may seem to be those of a serous meningitis or (when the exudation is circumscribed) of a cerebral tumour (*see* p. 326).

Treatment.—Up to the present there is no specific remedy for the disease, and treatment has mainly to be directed towards relief of symptoms and maintenance of nutrition. In selected cases lumbar puncture is sometimes followed by relief of head symptoms, and may be repeated as thought necessary, provided the tension of the cerebro-spinal fluid is raised.

CEREBRAL ABSCESS (SUPPURATIVE ENCEPHALITIS)

Etiology.—The source of the infection in a case of cerebral abscess may be in the skull or in some distant part of the body. Chronic suppuration of the middle ear is by far the most common cause, but infection may be carried equally well from other neighbouring parts, and Sir StClair Thomson has shown that diseases of the nasal sinuses are more important in this respect than had generally been supposed.

Necrosis of any of the bones of the skull, arising from injury, syphilis, or other diseases, may be followed by an abscess.

From more distant parts the infection is particularly apt to follow empyemata, bronchiectasis, and pulmonary abscess.

At other times the cerebral suppuration is but part of a generalized pyæmia, in which case the abscesses are often multiple.

The posterior parts of the temporo-sphenoidal lobes, and the cerebellum, from their proximity to the bones of the ear, are the areas in which abscesses most commonly occur; less often the occipital and parietal lobes are infected. When the infective matter is brought from distant parts the left side of the brain is rather more likely to be attacked than the right.

Pathology.—When the abscess follows diseased bone the dura mater over the necrosed area is usually thickened, but the superficial portion of the cortex generally appears healthy to the naked eye, and at a variable distance beneath its surface is situated the collection of pus. The channel by which the infection is conveyed to the brain is probably not always the same; sometimes it is by lymphatics, and sometimes by veins.

If the abscess has existed for some time, it is likely to be enclosed by a definite capsule, the result of organized products of inflammation in the neuroglial tissue; often, however, there is no such boundary, and a large area of brain tissue may be permeated by pus, like water in a sponge.

The colour of the pus is usually a greenish hue, and the odour is generally foul. Streptococci and staphylococci can often be found, as also, occasionally, may tubercle bacilli and other specific micro-organisms.

Symptoms.—The symptoms of an abscess, as might be expected, resemble to some extent those of tumour, and they may be divided in a similar fashion into *general* and *local* (p. 352).

Sometimes an abscess may exist for an indefinite time without producing any symptoms at all; and in chronic cases, *latent*, *acute*, and *terminal* stages may be well defined. In many there is a "latent period" of some length. When symptoms arise, headache, vomiting, and optic neuritis may all be present; but, compared with tumours, the pressure produced by an abscess is not often so great, and thus optic neuritis is not so frequently found, nor is it usually so intense, and for the same reason the vomiting and headache may be less troublesome. On the other hand, mental dullness is generally more pronounced than in the case of tumours. The slow pulse is present in both. In the terminal stages there may be signs of septic absorption which do not exist in most cases of tumour, e.g. a quickened pulse and an irregular temperature, with occasional rigors; and in the last stages of the disorder there may be delirium and coma. The onset of these more general symptoms is suggestive of the coincidence of meningitis.

In many cases the septic symptoms are rendered more intense by absorption of septic matter from other regions besides that of the actual abscess.

Left to itself, an abscess which is giving rise to acute symptoms will cause death, either by direct pressure on vital parts, or after rupture into one of the ventricles, or by infection of the membranes and the supervention of general meningitis.

The symptoms upon which the diagnosis of the position of the abscess depends are those which are discussed in dealing with the localization of tumours (p. 355). In the *temporo-sphenoidal lobe* there will be the likelihood of sensory aphasia, or, if the abscess is in the forepart of the lobe, there may be, as in the instance narrated elsewhere (p. 365), a disturbance of the sense of smell. If the pressure is considerable the motor fibres may suffer, and there will be signs of hemiplegia on the opposite side of the body.

When the pus is in the lateral lobe of the *cerebellum*, vertigo, nystagmus, paresis of the external rectus, with conjugate or skew deviation of the eyes, are among the prominent localizing symptoms. One of the greatest aids in the diagnosis of the site of the abscess is the position of the primary suppuration, to which the abscess is secondary; the two are nearly always in close proximity, and in the majority of the cases the primary seat of disease is connected with the ear.

Diagnosis.—The principal local conditions from which abscess has to be distinguished are *meningitis*, *suppuration in the mastoid cells*, and *thrombosis of cerebral sinuses*; the difficulty in discriminating between these several conditions is increased by the fact that they may all be present at the same time.

If the disease is confined to the mastoid processes, the pain, redness, tenderness, and other symptoms will be unaccompanied by any definite signs of implication of the brain. There will not be mental dullness, vomiting, or optic neuritis, nor will there be signs of pressure on any particular set of fibres. Moreover, the temperature will usually be raised instead of being subnormal; and rigors are common.

It is thus more often by a process of exclusion than otherwise that the diagnosis is made. In the same manner, careful consideration has to be given to the possibility of an abscess complicating sinus thrombosis, the symptoms of which are detailed in Chapter XLI., p. 409.

In meningitis the signs are more generalized, and the cranial nerves are more likely to suffer. The patient tends to assume a curled-up position, is irritable, shuns the light, and objects to being touched; convulsions may also be present. The diagnosis may be aided by performing lumbar puncture, for the cerebro-spina fluid is liable to contain micro-organisms in the case of meningitis but not in abscess. Leucocytosis of the blood may be present in both cases.

A change in the percussion note over the side of the head in which the abscess is situated has sometimes been observed. Tenderness over the skull in the vicinity of the abscess is another sign that has occasionally been noted.

Prognosis and treatment.—The prognosis in the case of a patient with a cerebral abscess must always be doubtful. When there are several abscesses which are merely a part of some general pyæmic condition the outlook is hopeless; but when there is only one abscess the final result depends upon the possibility of opening and efficiently draining it.

If this can be accomplished—as is, fortunately, often possible when the abscess arises from disease of the middle ear and the mastoid—the chances of recovery are, on the whole, fairly good. It is obvious that no medicinal treatment can give relief, and therefore, when once the diagnosis is established, there should be no time lost in resorting to surgical measures.

CHAPTER XXXVII

INTRACRANIAL TUMOURS

Varieties.—The growths that occur in the brain comprise infective granulomata, carcinomata, sarcomata, gliomata, benign tumours of various kinds, and cysts of simple and of parasitic origin.

With the exception of carcinomata, the growths tend to occur before rather than after middle life, and are frequently found in children. Of the infective granulomata, tuberculous and syphilitic growths are the most common; they are frequently multiple. Tuberculous growths are chiefly met with in the young; in them they show a predilection for the cerebellum, and may sometimes subside into quiescence.

Gummata nearly always arise from the cerebral membranes, and only rarely are found deep in the substance of the brain. The commonest positions in which to find them are the base and cortex of the cerebrum, the structures of the cerebellum being invaded comparatively seldom. Gummata are frequently multiple and accompanied by a certain degree of meningitis, which causes further damage to the nerve substance around.

Actinomycosis has occasionally been found within the skull.

Carcinomata generally occur in later life, and are always secondary to a growth elsewhere. According to Handley,* the cerebellum shows a special liability to be attacked.

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* "Cancer of the Breast," 1906.

Sarcomata are either primary or secondary, and may arise in any part of the brain.

Endotheliomata grow from the membranes of the brain. They differ considerably from one another in the intensity of their malignancy.

The *psammomata,* which contain earthy matter and cholesterin, belong to this last group;

Gliomata originate in the neuroglia. They infiltrate rather than compress the brain tissue. Owing to their close resemblance to the cerebral substance their boundaries are difficult to define, and their general appearance is often one of hypertrophy of a part of the brain. These tumours are usually single, and do not give rise to secondary deposits; they are prone to undergo degeneration, and occasionally they become quiescent.

The *benign tumours* are of comparatively rare occurrence.

Neuro-fibromata occasionally grow from the sheaths of the cranial nerves. *Adenomata* and *cystic degenerations* may arise in connection with the pituitary body.

Cholesteatomata, *lipomata,* and *angiomata* occur occasionally, and sometimes an *osteoma* grows inwards and compresses the brain.

Parasitic cysts occur in connection with the *Tænia echinococcus* and *Cysticercus cellulosæ*, and simple cysts are also sometimes found.

An intracranial *aneurysm* may give rise to symptoms of a tumour, according to its size and position.

Symptoms.—There are certain symptoms which may be associated with any intracranial tumour, irrespective of its nature or situation, while others are peculiar —or accidental, so to speak—to the position in which the tumour happens to be placed. Thus the symptoms may be grouped under the headings of *general* and *local*.

The **general** symptoms are mainly the result of increased intracranial pressure, and they include headache, vertigo, vomiting, optic neuritis, slow pulse, subnormal temperature, with possibly a general convulsion, and some mental failure.

It is hardly necessary to say that frequently not all of these are present in any given case, and that the absence of one or more of them by no means necessarily invalidates the diagnosis.

Headache.—Headache is one of the most frequent symptoms of intracranial tumours. After it has once set in it seldom disappears, but its intensity is nevertheless liable to considerable fluctuations. The usual history is that of a dull ache with agonizing exacerbations. When the growth is due to syphilis the exacerbations are most notable during the night, and there is often some localized tenderness, which can seldom be satisfactorily demonstrated in association with other kinds of tumour.

With the possible exception of the syphilitic cases, the position of the headache cannot in itself be relied upon to any extent as a guide to the position of the tumour, though, in conjunction with other symptoms, the seat of the pain may be strongly suggestive.

In cerebellar tumours, for example, the pain is often felt most intensely in the occipital region, and, if the other signs are in keeping, this localization of pain may be given a certain value; but it sometimes happens that the headache of tumours in the posterior part of the brain is chiefly frontal in distribution, and, conversely, that of frontal tumours may be mainly felt at the back of the head.

Vomiting.—Vomiting is a common symptom. Sometimes it is preceded by nausea, while at other times the contents of the stomach are suddenly brought up without any warning. It may occur without any definite relation to taking food. Vomiting is liable to be very pronounced in tumours situated beneath the tentorium, where their growth is confined and causes a rapid rise of pressure.

Optic neuritis.—Of all the signs of intracranial tumour, optic neuritis is probably the most important. It occurs in a large percentage of cases, and, like the vomiting, is apt to be very intense with subtentorial growths. The neuritis is sometimes more advanced on one side than the other, and such inequality has a certain degree of value in localization by suggesting that the growth is in that half of the brain which corresponds to the inflammation of longer standing. Too much dependence must not, however, be placed upon it.*

It may also be said, roughly speaking, that the rapidity of the neuritis tends to be somewhat proportionate to the rate of growth of the tumour, though there must necessarily be many exceptions to this statement.

Temporary attacks of dimness or loss of vision, which are usually accompanied by exacerbations of headache and vertigo, are occasionally met with during the course of intracranial tumours, more especially of those of the cerebellum, and are regarded as being due to a temporary strangulation of the optic nerve or to pressure of the infundibulum upon the optic chiasma (Leslie Paton).

The *slow pulse* and the *low temperature* are both variable symptoms, and are produced by the increased intracranial pressure.

General convulsions, though important when they occur as an early symptom, are distinctly uncommon, but, unless their possible connection with tumours be remembered, they may easily be diagnosed as epilepsy until suspicions are aroused by the appearance of other signs. The greatest safeguard against overlooking a tumour consists in systematically examining the optic discs.

Mental changes sometimes occur quite apart from the disturbance of any special part of the brain, and are then to be attributed chiefly to the rise of pressure within the skull.

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* Of 50 cases analysed by C. E. Beevor, in 19 the neuritis was more intense on the side of the lesion, in 11 it was more marked on the side opposite the lesion, and in 13 it was equal on the two sides. In the remaining 7 there was no neuritis.

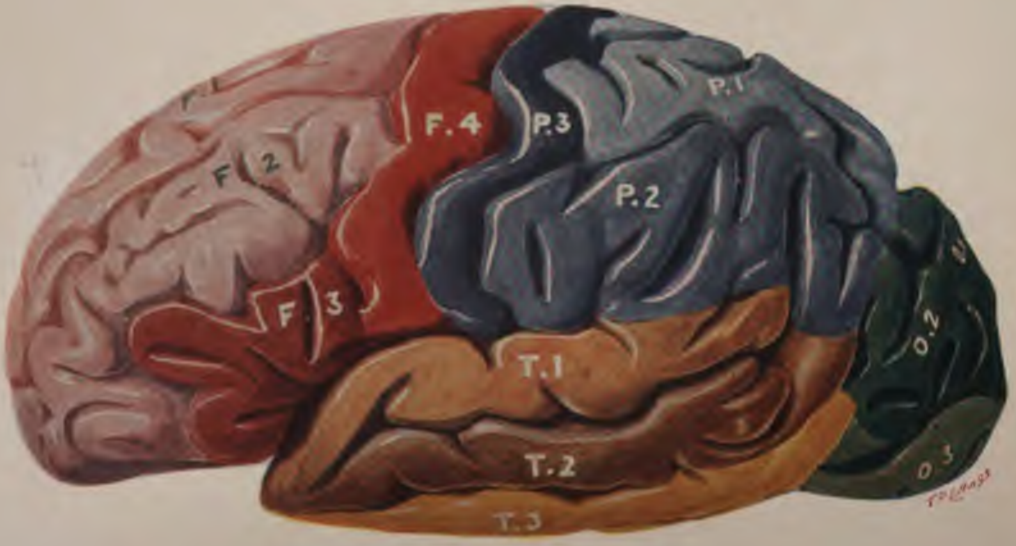

PLATE 18.—Diagram of the convolutions of the cerebral cortex.

Local symptoms are considered in the following section :—

LOCALIZATION OF INTRACRANIAL TUMOURS

The ability to localize the position of intracranial tumours depends first upon a knowledge of the anatomical seat of those functions of the brain which are disturbed. Hence it is only possible to localize tumours accurately when they are situated in certain regions of the brain, for when they are in parts whose functions are unknown—the so-called "silent areas" —the general symptoms are accompanied by no special local disturbances by which the position of the tumour can be defined.

Another source of difficulty lies in separating the symptoms produced in the immediate vicinity of the tumour from those which arise some distance off as the result of indirect pressure effects.

The value of local symptoms also varies according to the date of their appearance. They are most reliable when their advent coincides with or shortly follows the appearance of the general symptoms, for when local symptoms appear only a long time after the onset of general symptoms, the chances are many (as shown by Collier) that they may not then point to the primary position of the growth, but rather represent the results of such secondary changes as hydrocephalus, meningitis, vascular lesions, and metastatic deposits ; and, further, as the same author has also shown, the long-continued absence of local signs, when the general signs are well marked, is in itself of importance in suggesting that the tumour is situated above rather than below the tentorium, since in the latter case localizing symptoms generally appear early.

Assuming that the signs of a tumour are present, the various effects, according to the position of the growth, may now be considered.

1. **Tumours interfering with the motor tracts.** —When the presence of a tumour is associated with loss

of voluntary movements we may assume that the fibres which conduct the motor impulses are interrupted at some point in their course between the motor area of the cerebral cortex and the decussation at the medulla, and the different effects may be considered according as the growth is situated in (i.) the motor area of the cortex, (ii.) the region of the internal capsule, (iii.) the crus cerebri, or (iv.) the pons Varolii.

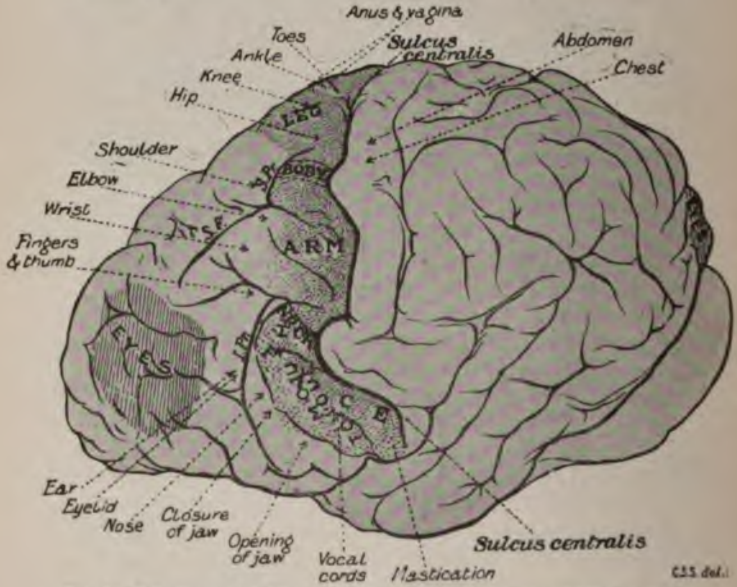

Fig. 90.—Diagram showing the situation of the motor areas in the cortex. (*Sherrington and Grünbaum.*)

i. **Tumours of the motor area of the cortex.**—The extent of the motor area of the cortex is seen from Fig. 90, which depicts the arrangement found by Sherrington and Grünbaum in the highest apes, and which presumably closely conforms to that of the human brain. It will be noticed that there are two important modifications of the plan which was formerly considered correct, viz.:

(*a*) The whole of the motor area is situated in front of the fissure of Rolando and is confined to the ascending frontal convolution.

(*b*) The representation for movements of the trunk is placed between those for the arm and leg, instead of being situated on the margin of the great longitudinal fissure.

Now, if a tumour be situated in this motor area, it is obvious that at first it will be likely to affect only a small portion of the convolution, and, as a general rule, the weakness is for a time confined to one limb or to the face, i.e. there is a *monoplegia*, usually of slow development, which later on extends to a *hemiplegia* as the growing edges of the tumour spread into the neighbouring areas. At the same time that the growth is reducing the power of movement, it is also irritating the motor cells in its immediate vicinity. The result of this irritation is seen in convulsions, which begin in a group of muscles corresponding to the irritated area of the cortex, and subsequently spread with some deliberation to other parts of the limb, when they may either subside without loss of consciousness or develop into a general convulsion. The significance of these localized convulsions as the indicators of the particular area of cortex that is diseased was first pointed out by Hughlings Jackson, from which circumstance they are known as *Jacksonian epilepsy*.

Localized convulsions are not in themselves pathognomonic of the presence of gross cerebral lesions, for they are frequently met with in the course of ordinary epilepsy. Their significance is the same in both cases, inasmuch as they indicate the part of the brain in which the epileptic discharge begins; but, where the general signs point to the presence of a gross lesion, their occurrence is of the greatest importance in enabling its position to be determined. Lastly, in addition to loss of power, a tumour of the motor cortex is sometimes accompanied by some disturbance of sensation, due probably to pressure on the ascending parietal lobe. The sensory

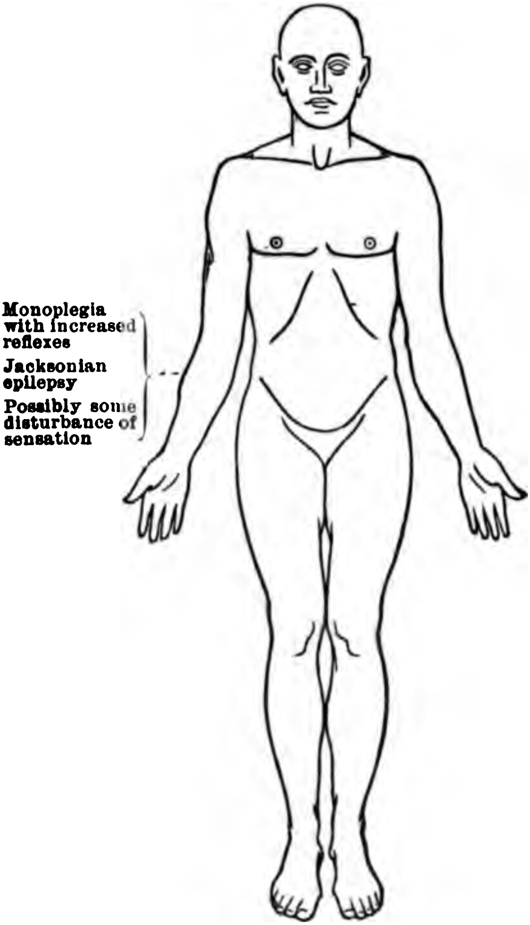
Monoplegia with increased reflexes
Jacksonian epilepsy
Possibly some disturbance of sensation

Fig. 91.—Scheme of the principal localizing symptoms produced by a tumour in the cortical motor area for the arm.

disturbance from a cortical lesion will not be in the form of loss of crude recognition of touch, pain, heat and cold,

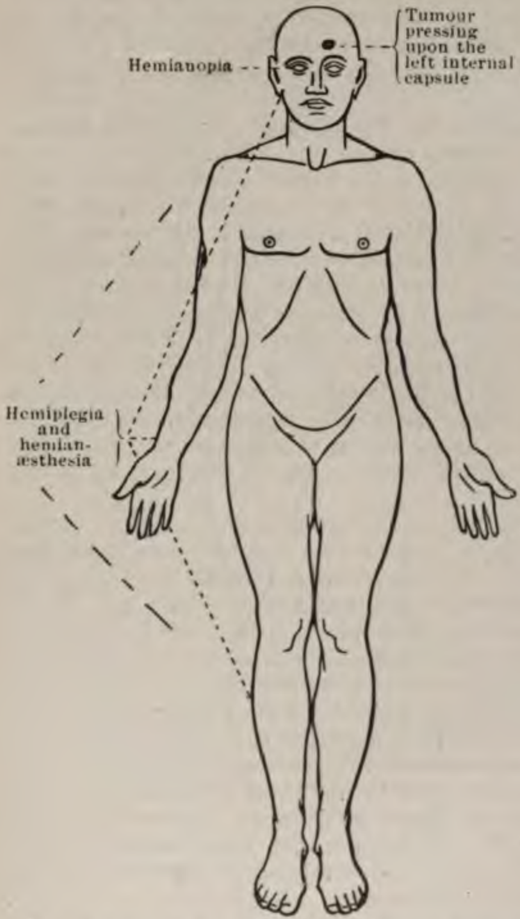
Tumour pressing upon the left internal capsule
Hemianopia
Hemiplegia and hemianæsthesia

Fig. 92.—Scheme of the principal localizing symptoms produced by a tumour pressing on the internal capsule.

but will show itself by want of discriminative power as described under Tumours of the Parietal Region (p. 363). To sum up, the main signs upon which reliance can be placed in the localization of a tumour in the area of the motor cortex are *monoplegia* and *Jacksonian epilepsy,* with perhaps some *disturbance of sensation.* (Fig. 91.)

ii. **Tumours in the region of the internal capsule.**—At the internal capsule the motor fibres are packed closely together, and a tumour pressing upon them is most likely to impair the functions of all, and so to cause a hemiplegia of slow onset. At the same time there may be a certain amount of hemianæsthesia, since the sensory fibres occupy the posterior third of the posterior limb of the capsule, but in slow-growing tumours this is often not noticeable. The fibres of vision from the occipital lobes may also be interrupted as they pass across the hindmost part of the internal capsule, in which case there will be hemianopia. There will be no Jacksonian convulsions, though there may occasionally be a generalized fit due to the effects of general pressure on the cortex.

The main localizing symptoms of a tumour pressing on the internal capsule are, therefore, *hemiplegia, hemianæsthesia,* and *hemianopia.* (Fig. 92.)

iii. **Tumours of the crus cerebri.**—There will be the same tendency for a tumour of the crus to give rise to hemiplegia and hemianæsthesia of the opposite side as there is in the case of a tumour situated in the internal capsule, with the probable addition of paresis of certain cranial nerves, more especially of the 3rd, from pressure on its nucleus or on the fibres springing directly from it. This gives rise to a "crossed" paralysis, i.e. the face, arm, and leg are paralysed on the one side (the side opposite the tumour), and the ocular muscles supplied by the 3rd nerve are paralysed on the other side (the same side as the tumour).

In addition, other cranial nerves, e.g. the 4th, 5th, and 6th, may be pressed upon if the tumour is a large one.

Tremors have also been observed in these cases.

Thus the main diagnostic points of a tumour in the crus cerebri are *crossed paralysis* (i.e. loss of power in the face, arm, and leg on the side opposite to the tumour, and paralysis of ocular muscles, and possibly facial muscles, on the same side), *hemianæsthesia* and possibly *tremors.*

iv. **Tumours in the pons.**—Tumours which are situated in the pons tend to cause symptoms somewhat similar to those of the crus, inasmuch as they give rise to hemiplegia and hemianæsthesia of the opposite side and also to a crossed paralysis (Figs. 93 and 94). In this instance it is the facial nerve that usually suffers on the same side as the tumour, but the oculomotor (3rd), the abducens (6th), and the auditory (8th) may also be affected. It is also in a similar manner possible for lesions of the pons to cause *crossed hemianæsthesia,* by damaging the fibres of the trigeminal nerve going to the one side, and those for the arm, trunk, and leg on the opposite side. There may be conjugate deviation of the eyes away from the side of the tumour, or nystagmus may be present. Optic neuritis is often comparatively late in

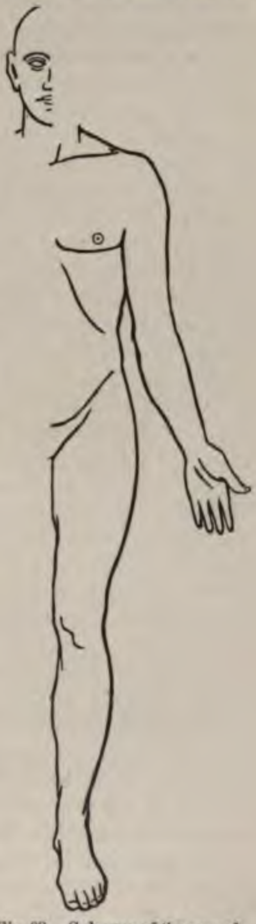

Fig. 93.—Scheme of "crossed paralysis: face affected on one side, arm and leg on the other.

making its appearance. The main points to be relied on are *hemiplegia* and *hemianæsthesia* on the opposite side, with *facial paralysis*, *deafness*, and *ocular palsies* on the same side as the tumour.

In the *medulla* the motor fibres of the two sides are so close together that a tumour in their immediate neighbourhood is liable to cause bilateral paralysis, though in all probability the loss of power will be more marked on one side than on the other.

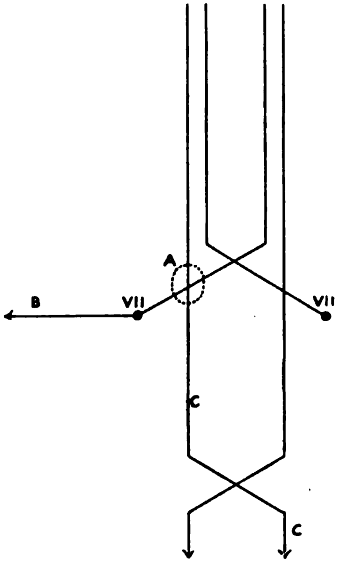

Fig. 94.—Diagram to show the mechanism of crossed paralysis. The lesion at A paralyses the facial nerve, B, on the one side and the fibres for the arm and leg, C, on the other.

In addition to this, there is every likelihood of the existence of symptoms due to pressure on the bulbar nuclei, such as difficulty in speaking and swallowing, and weakness of the tongue. Glycosuria may also be found.

2. **Tumours situated in parts of the cerebral cortex other than the motor area.** i. **Tumours of the frontal lobes.**—Tumours of the frontal lobes, whether cortical or subcortical, are admittedly difficult to diagnose, since they may attain to a large size and yet give rise to no localizing symptoms.

The functions of the frontal lobes are regarded as being mainly connected with the intellect, and hence mental disturbance, in conjunction with the absence of signs that the tumour is situated elsewhere, is the chief condition upon which reliance must be placed. Mental disturbances are, however, often caused by tumours in other parts of the brain, although Mills, who has contributed largely to this important subject, considers that the conflict of evidence is more apparent than real, and that it would disappear if sufficient care were taken to analyse the symptoms and to exclude those which are of a more general character and obviously due to widespread irritation from a tumour. Mills believes that if every case of disease of the prefrontal lobes (and more especially of the left lobe) were carefully analysed, some mental symptoms of a special character in connection with the higher psychic activities would be recognized.

Grainger Stewart has observed that the superficial abdominal reflexes are often diminished or lost on the opposite side to the tumour, and that there is apt to be a fine, rapid, vibratory tremor of the limbs on the same side as the growth.

As the tumour grows, the signs of encroachment on the motor areas for the head and eyes, the limbs, or the motor centre for speech (Broca's convolution) may give the clue to its position.

ii. **Tumours of the parietal region.**—A large number of sensory fibres impinge on the parietal lobes, and a disturbed function of these is the chief effect of a tumour of this region.

The loss of sensation in these cortical cases, as already mentioned (p. 359), will not take the form of inability to recognize crude sensations of touch, pain, heat and cold, but will be characterized by lack of discrimination as regards (1) recognition of spatial relations, (2) graduated responses to stimuli of different intensity, (3) appreciation of similarity and difference in external objects brought into contact with the surface of the body. The form of discrimination most often impaired is that of knowledge of spatial relations, and clinically an inability to recognize passive movements of an affected limb is to be regarded as one of the principal signs of disease of the sensory area of the cortex (Head). Additional signs of the same nature are loss of power to distinguish differences in weights and shapes, the last-named being often spoken of as the "stereognostic sense" (*see* Sensation and the Cerebral Cortex, p. 23). To the sensory signs others are usually added through the encroachment of the tumour on the parts around. Thus, if it extends forwards, there will be motor symptoms; if backwards, the functions of the angular gyrus and occipital lobe will be impaired; while if the disease spreads beneath the cortex, it may affect the motor fibres as they pass through the corona radiata. Convulsions associated with tumours of the parietal lobes are usually preceded by sensory warnings.

When the tumour encroaches on the occipital lobe there may be hemianopia. Visual aphasia (word-blindness) may result from tumours of this region when they are situated on the left side. Fits beginning with visual warnings may also be associated with tumours encroaching on the occipital region.

iii. **Tumours of the occipital lobes.**—Fits beginning with visual warnings may be the early signs of tumours of the occipital region of the cortex. There may also be hemianopia, i.e. loss of half-vision in the corresponding parts of the two fields (homonymous or simple hemianopia); but the value of this symptom is, of course, frequently lost through the general impairment of vision from optic neuritis. Occasionally only a quadrant of the usual field is lost (quadrantic hemianopia). If the tumour is on the left side there may be word-blindness with diminished fields of vision, as mentioned in the previous paragraph.

iv. **Tumours of the temporo-sphenoidal lobes.**—Tumours of the temporo-sphenoidal lobes may cause deafness on the opposite side, and, if situated on the left side, there may also be sensory aphasia (word-deafness). Auditory warnings may occur before a fit, or hallucinations of hearing may be present. Extension of the tumours inwards may cause pressure on the motor and sensory fibres for the opposite limbs.

v. **Tumours of the uncinate gyrus.**—Tumours of the anterior part of the temporo-sphenoidal lobe are sometimes accompanied by the "dreamy states" first described by Hughlings Jackson.

Sensations of smell may also accompany tumours in this region—a fact which supports the view that the cortical area concerned with olfactory sensations is that of the uncinate gyrus or its immediate neighbourhood.

One such case which the author had an opportunity of observing was that of a young man under the care of Sir James Kingston Fowler * at the Middlesex Hospital. Along with general signs of cerebral abscess there were frequent attacks of epileptic vertigo, which were preceded by a sense of an unpleasant smell. After death a large abscess was found in the fore part of the temporo-sphenoidal lobe.

Another important fact connected with this case was the excessive hunger and thirst from which the patient suffered, a condition which is occasionally found in connection with injuries and diseases of various parts of the brain, but more especially, it would appear, when the lesion is in the locality of the temporo-sphenoidal lobe, a point which has been brought out by Stephen Paget † from a survey of 14 cases of the kind which he collected:

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* *See* "Post-mortem Records of the Middlesex Hospital," 1895.

† *Trans. of the Clin. Soc. of London*, 1897.

Thus, *sensations of smell*, together with "*dreamy mental states*," and perhaps *voracious hunger* and *thirst*, are the signs which suggest that a tumour is situated in the neighbourhood of the uncinate convolution.

3. **Tumours of the optic thalamus.**—It is often very difficult to locate tumours of the optic thalamus. Most of the symptoms are due to pressure on the internal capsule, and therefore consist of hemiplegia, hemianæsthesia, and hemianopia. Loss of control over the emotional movements, the early appearance of hemianæsthesia and athetoid movements, are some of the signs that are most likely to occur.

4. **Tumours of the corpora quadrigemina.**—In tumours of this region there is generally well-marked paralysis of the eye muscles, probably partly produced by pressure on the nuclei of the 3rd nerves. The reaction of the pupils both to light and to accommodation is often impaired, and nystagmus may occasionally be observed. There is also difficulty in maintaining the equilibrium and a tendency to fall in one direction.

5. **Tumours of the corpus callosum.**—It is but seldom that a growth beginning in the corpus callosum can be localized with any degree of certainty. As a rule, the general symptoms develop slowly, and no localizing signs occur until the tumour has invaded one or other hemisphere sufficiently far to injure the motor fibres. In some cases mental changes have been observed, suggesting a lesion of the frontal lobes.

6. **Tumours at the base of the brain.**—Tumours at the base of the brain are generally accompanied by early signs of palsy of cranial nerves, the situation of which is depicted in Plate 19.

i. In tumours of the **pituitary body** the optic chiasma is very apt to suffer, and if it be compressed at its centre a double temporal hemianopia will ensue, followed by more complete loss of vision as the pressure extends to the fibres which constitute the temporal portions of the optic nerves (*see* Fig. 17, p. 85). These symptoms

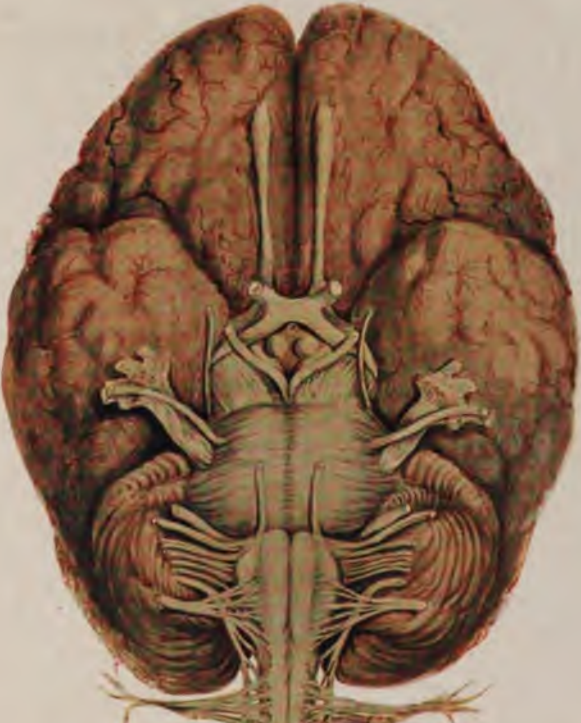

PLATE 19.—The cranial nerves at the base of the brain.
(From a painting by Sir Charles Bell at the Middlesex Hospital Medical School.)

are often the first complained of by patients suffering from *acromegaly*, the characters of which are described at p. 378.

Tumours growing in the middle fossa generally paralyse the ocular and trigeminal nerves, while those of the posterior fossa affect the 6th nerves very early, and signs of pressure on the crus, pons, and cerebellum may appear.

ii. **Tumours at the angle of the pons and cerebellum.**—Tumours growing at the angle of the pons and cerebellum are of special interest from the relative frequency of their occurrence and the possibility of their removal. Although this region of the brain is not exempt from tumours of every kind, the one that is especially prone to arise is of a nodulated fibrous appearance, and not infrequently attains the size of a walnut or a pigeon's egg.

These growths have been variously described as fibro-neuromata and sarcomata, and often appear to arise from the sheath of the auditory nerves. Bland-Sutton looks upon some of them as psammomata, and believes that they have originated from processes of the chorionic villi of the fourth ventricle. Sometimes they are bilateral.

These tumours produce localizing symptoms from pressure—

(1) On the *cranial nerves* in their immediate vicinity.

(2) On the *cerebellum*.

(3) On the *pons* and *medulla*.

Reference to Plate 20 will make these three lines of pressure clear.

(1) *Pressure on the cranial nerves.*—The nerves most likely to suffer are the 5th, 6th, 7th, and 8th, and, as the tumour generally arises in close apposition to the auditory nerve, *deafness* is often one of the earliest symptoms.

The *facial trunk* is generally compressed at the same time, but it is apparently more capable of resisting pressure than the auditory nerve, so that the facial muscles often suffer to a surprisingly slight extent.

them is only very slowly increased. The main points to be borne in mind for localizing tumours in this situation are—

(*a*) Pressure on the *auditory* (chiefly), facial, abducens (6th), and trigeminal (5th) nerves on the *same side* as the tumour.

(*b*) Symptoms of disease of the lateral lobe of the *cerebellum*, also on the *same side*.

(*c*) *Weakness* of arm and leg with increased deep reflexes and *extensor* type of plantar reflex on the *opposite side*.

Since these tumours usually press upon three different sets of structures, namely, cranial nerves, cerebellum, pons and medulla, it is obvious that difficulty must sometimes be experienced in deciding which of the three parts is the primary site of the growth. In other words, is the growth situated in the angle between the pons and cerebellum, in the cerebellum, or in the pons?

Early signs of implication of the cranial nerves—as, for instance, when deafness, along with weakness of the facial muscles and noises in the ear, makes its appearance slowly and gradually, before anything else is noticed—are strong evidence that the tumour is in the pontine-cerebellar angle. In cases of primary intracerebellar growth, these cranial nerves, with the exception of the 6th, would not be likely to suffer till other symptoms were well developed. Grainger Stewart and Gordon Holmes have observed that, in the vertigo accompanying tumour of the lateral lobe of the cerebellum, the direction in which the patient feels himself to be rotating is away from the side of the lesion; while in cases of extracerebellar tumour the sensation of turning is towards the side of the lesion.* The chief effects of tumours of the pons have already been considered, and the prominence of "crossed paralysis," conjugate deviation of the eyes, and paresis (sometimes bilateral) will generally serve to distinguish pontine tumours from those now under consideration.

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* *Brain*, Part cviii.

7. Tumours of the cerebellum.—In tumours of the cerebellum the general symptoms of an intracranial growth are, as a rule, exceptionally well marked.

Optic neuritis is more frequently met with in association with these tumours than with those of any other part of the brain. It comes on early, and the inflammatory process is apt to be more advanced in the eye of the same side as the growth. *Headache* is usually very intense, and often referred to one or other side of the occipital region; there may also be rigidity of the neck muscles. *Vertigo* and *vomiting* are likewise generally prominent symptoms and appear at an early date.

The influence of each half of the cerebellum is chiefly exerted on the same side of the body, in contradistinction to that of the cerebrum, where each half mainly governs the opposite side; tumours and other diseases of the cerebellum therefore tend to produce symptoms on the same side of the body as the lesion.

The signs of a cerebellar tumour may be divided into two groups, according as they depend directly upon the disordered function of the cerebellum, or indirectly upon pressure on neighbouring parts.

(1) **Direct effects of cerebellar lesions.** *Motor symptoms.*—These are generally *weakness* and *want of tone* in the limbs on the same side as the tumour. This loss of power, often slight, on the same side as the lesion, and due directly to deficiency of cerebellar function, must not be confused with paresis on the opposite side, which may arise from the effects of pressure on the medulla or pons.

Incoördination, usually more prominent on the side of the lesion, is especially marked in the lower limbs, but is not usually increased when the eyes are closed. One effect of incoördination of the upper limbs which is sometimes seen is an inability to pronate and supinate the forearm rapidly.

The *gait* is of a reeling character, and the legs are kept wide apart; there is also a tendency to turn and fall to one side, usually, though not always, towards the side of the tumour.

Compensatory attitudes are sometimes assumed in attempting to neutralize the tendency to incline towards the diseased side. These are often excessive, and cause the patient to incline unduly towards the sound side.

Tremors are often present.

Tonic spasms, causing opisthotonos and rigidity of the whole body, have sometimes been noted, but they are uncommon. Occasionally there are jerky movements, and, unless care be taken to inquire closely into the character of such attacks, a patient's description of the condition may suggest some similarity to a Jacksonian fit.

Position of head.—It has been shown that in a certain number of cases the head is tilted so that the ear approximates to the shoulder on the side opposite the lesion, while the face is turned up to the side of the lesion.

Forced movements, in which the patient rotates round the long axis of his body, sometimes occur.

Ocular movements.—*Nystagmus* is very frequently present. Sometimes it is lateral, and brought out by fixing the eyes to one or other side; at other times it is associated with all the movements of the eyeball, and it may be of a rotary kind. The movements are often of different character, according to whether the eyes are turned to the right or left, being short and sharp in the one instance, and longer and slower in the other. In such cases the more deliberate movements generally occur on the same side as the tumour.

Skew deviation, where one eye is turned downwards and inwards and the other upwards and outwards, is sometimes observed. In a case of cerebellar abscess under the care of the author, both eyes were turned downwards and inwards.

Weakness of the external rectus, which is so frequent in cerebellar disease, is due to pressure on the 6th nerve, and reference is made to this in connection with the indirect results of these tumours (*see* below).

Vertigo is generally a prominent symptom and occurs in paroxysms.

Reflexes.—The knee-jerks vary in different cases; if they are unequal, the more active, according to experimental results, is likely to be on the side of the lesion (Risien Russell).

In tumours of the *middle lobe* there may be retraction of the head, arching of the back, and extension of the legs (cerebellar attitude of Hughlings Jackson), which position may be further intensified by the occurrence of tetanus-like seizures. There is generally a tendency to fall backwards.

(2) **Indirect effects of cerebellar lesions.**—Of the symptoms produced by pressure on the parts around, the most frequent is a paralysis of the 6th nerve on the same side as the lesion, which leads to weakness of the external rectus and subsequently to a tendency for the eyes to deviate from the affected side. Other cranial nerves, such as the auditory, facial, and trigeminal, may be injured, though, as a rule, much less severely in cerebellar tumours than in those which arise at the angle of the cerebellum and pons.

Pressure on the pons may cause crossed paralysis, i.e. hemiplegic symptoms on the opposite side of the body, with ocular paralysis on the same side; while pressure on the medulla may produce pharyngeal and laryngeal symptoms. Attacks of dyspnœa are also apt to occur from pressure on the medulla, and may be a cause of sudden death.

When the tumour grows slowly it may cause hydrocephalus, the symptoms of which may mask those produced directly by the primary disease.

Diagnosis of intracranial tumours.—The diagnosis of intracranial tumour is made from the general signs of a growth, aided by the local ones if present.

Mistakes are most likely to arise when headache and vomiting and perhaps optic neuritis are present from other causes, such as severe *anæmia*, chronic *lead poisoning*, and especially *Bright's disease* with albuminuric retinitis (*see* Plate 5, Fig. 3, facing p. 82). Where the symptoms are very obscure a case may even for a time be regarded as functional.

Having decided that an intracranial tumour is present, it is necessary to try (1) to locate its position, (2) to infer its probable structure, and (3) to decide whether any attempt shall be made to remove it.

Prognosis and treatment.—The prognosis must depend upon the conclusions that are arrived at concerning (1) the nature of the tumour, (2) the probability of its removal by medical or operative measures, and (3) the possibility of the occurrence of spontaneous retrogression. A benign tumour is, other things being equal, obviously more hopeful than one that is malignant, and should the latter be a secondary deposit the outlook will in most cases naturally be hopeless. It is in syphilitic growths that most is to be expected from medicinal measures, and when there is the least chance of the disease being of this nature a thorough course of treatment should be given. Tuberculous deposits often show a tendency to retrogress under the influence of cod-liver oil, iron, good food, and fresh air.

With these exceptions no reliance can be placed on drugs, and, if no benefit be obtained from a thorough but brief trial of treatment, the desirability of an attempt to remove the tumour must be seriously considered. The decision must depend chiefly on the position and apparent accessibility of the growth and on the probable effects of its attempted removal.

Tumours of the frontal, parietal, occipital, and temporo-sphenoidal cortex can all be reached, as also can those of the cerebellum and cerebello-pontine angle.

While the danger from an operation is in all cases considerable, it differs according to the position of the tumour, being less when the growth is situated in the mid- and fore-part of the brain, and greater when it is in the region of the cerebellum. Thus, although a tumour of the lateral recess (cerebello-pontine angle) may be easily shelled out of its bed, the shock produced in reaching it is great enough to make the mortality a very high one.

Where removal of the tumour, owing to its size, nature, or position, cannot be accomplished, an endeavour should be made to relieve the symptoms and prolong life by the operation of *decompression*, i.e. by removal of a large piece of bone and (usually) incision of the dura mater. By this means the intracranial pressure is lowered, the beneficial result of which is especially seen in the consequent tendency of the optic neuritis to subside—a matter of the greatest importance in those cases where the growth of the tumour eventually becomes stationary. Apart, therefore, from other considerations, the operation of decompression is a valuable means of preserving the sight. Care must be taken not to allow the changes in the retina to proceed too far before something is done, and, during the period that medicinal treatment is being carried out, careful observations must frequently be made on the condition of the optic discs and on the fields of vision, for any rapid contraction of the latter indicates the necessity of relieving the pressure without further delay.

The smaller degree of shock which follows the operation of decompression makes it a far less dangerous procedure than that of removal or attempted removal of the tumour, and, considering the high mortality and the disappointing results that follow the latter operation, it is well to be content with decompression unless the circumstances are unusually favourable to the total removal of the growth.

CHAPTER XXXVIII

DISEASES OF THE PITUITARY BODY

The pituitary gland is situated in the sella turcica of the sphenoid bone, and consists of an anterior and a posterior lobe. (Fig. 95.)

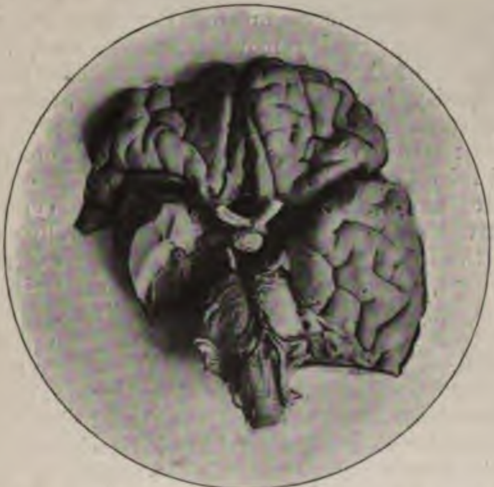

Fig. 95.—Base of brain showing position of pituitary body just behind the optic commissure.

The anterior lobe arises as an outgrowth of the pharynx, and is composed of glandular tissue which bears considerable similarity in structure to that of the thyroid body. The posterior lobe arises from the mid-brain, to which it remains attached by a stalk known as the infundibulum, and is composed chiefly of loosely arranged neuroglial tissue, together with pigmented cells and blood-vessels. Between these two lobes there is a layer known as the pars intermedia, the structure of which is partly glandular and partly vascular.

The secretion of the posterior lobe exerts an influence to some extent similar to that of the secretion of the suprarenal body, inasmuch as it causes a rise of blood-pressure, but its chief function seems to be concerned with carbohydrate metabolism. Over-activity of this lobe is apt to be associated with glycosuria, while diminished secretion is accompanied by a tolerance of carbohydrates and a consequent tendency to obesity.

Cushing's experiments on adult dogs show that when the pituitary gland is removed, with the exception of a fragment of the anterior lobe, the resulting changes are adiposity, increased toleration of sugar, subnormal temperature, and reversive sexual changes, while in puppies retarded skeletal growth, psychic disorder, and sexual infantilism occur.

Cases are met with clinically in which symptoms similar to these are present. They are generally classed under the headings of Frölich's disease, or dystrophia adiposo-genitalis (Fig. 96), and are characterized by adiposity with skeletal and sexual infantilism. In such instances it may usually be inferred that the posterior lobe of the pituitary body is at fault, but when making that diagnosis it must not be forgotten that abnormalities of the suprarenal and the sexual glands, with which the function of the pituitary is closely correlated, may also be partly responsible for the condition.

The anterior lobe of the pituitary body is closely concerned with skeletal growth, and when its function is disturbed on the side of excessive secretion (hyperpituitarism) an overgrowth of the skeletal structures takes place which, according to circumstances, results in the production of giantism or of acromegaly.

If the disease occurs while the subject is still young and before ossification of the epiphyses has taken place, the result is giantism, while in older people the growth takes place in other directions, with the resulting disease

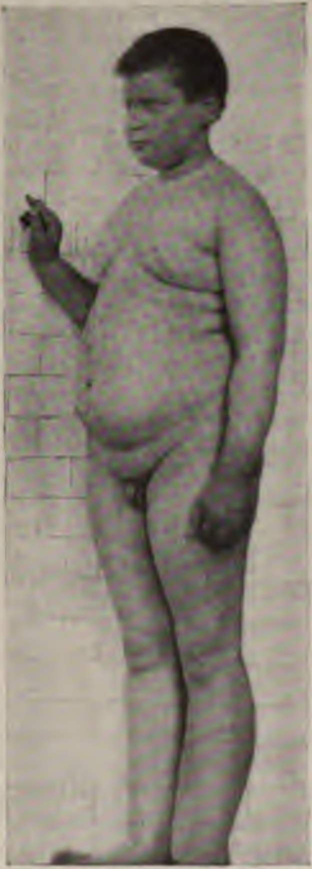

Fig. 96.—Dystrophia adiposo-genitalis in a boy aged 11. (*Case under the care of Dr. H. G. Turney.*)

known as acromegaly. There is thus a possibility of the symptoms beginning with those of giantism while the bones are capable of growing longer, and ending with those of acromegaly when age has rendered an increase in length no longer possible.

The classification of cases of pituitary disease into states of over-activity (hyperpituitarism) and under-activity (hypopituitarism) must not be taken too rigidly, for Cushing has shown that it is common to find a phase of over-secretion followed later by one in which secretion is diminished, and in such a case symptoms of the later stage of deficiency may be added to those of the earlier one of over-activity. Hence Cushing considers that in the majority of cases the term "**dyspituitarism**" is the more appropriate, and he suggests a classification as follows:

1. Cases in which symptoms of hyperpituitarism predominate, producing either giantism or acromegaly, according to the age at which the patient is affected.
2. Cases in which the symptoms of hypopituitarism predominate, with the consequent presence of adiposity and sexual infantilism.
3. Cases in which the symptoms exhibit features of both states.

ACROMEGALY

Acromegaly was first fully described by Pierre Marie, who associated the condition with enlargement of the pituitary gland. Subsequent observations showed that enlargement does not always occur, and for a time there was considerable doubt as to the relationship of the gland to the symptoms, until it became recognized that abnormalities in the quantity and probably also in the quality of the secretion can occur without enlargement of the gland necessarily taking place.

Symptoms and morbid anatomy.—The symptoms of acromegaly may be divided into two groups:

(1) those directly due to enlargement of the gland, (2) those due to changes in general metabolism.

One of the most important local results of an enlarged pituitary body is a disturbance of vision, generally attributed to pressure on the optic chiasma. The most striking form which this disturbance takes is the development of bitemporal hemianopia, followed later by extension of the blindness into the nasal fields; and it is interesting to note that in these cases perimetric observations show further differentiation in that a piece of the field in the upper and outer part of the temporal half is generally obliterated before the rest.

The explanation usually given for this sequence of events is an anatomical one, which depends upon the arrangement of the optic fibres at the chiasma. Reference to Fig. 17 (p. 85) shows that the fibres from the inner half of the retina which correspond to the temporal field of vision decussate with their fellows of the opposite side, while those from the outer part of the retina which correspond to the nasal fields of vision pass backwards from the outer side of the retina without crossing. Thus any pressure on the centre of the chiasma such as might be expected to be exerted by an enlarging pituitary gland would first of all tend to press upon the crossing fibres and produce bitemporal hemianopia, while later on it would interfere with the outer fibres and so affect the nasal fields. On the other hand, when the pressure was unequal from the beginning, the failure of the fields would not show any definite division into temporal and nasal halves. The difficulty of accepting this explanation in its entirety lies in the fact that the size of the whole chiasma is so small that it is not easy to see how pressure can be limited to the crossing fibres for any length of time. It may be that, given equal pressure on all the fibres, the function of some fails before that of others. There are examples of this in other nervous structures, such as, for instance, in the case of the recurrent laryngeal nerve as described at p. 110. However this may be, a temporal hemianopia, followed later by pallor of the discs, is a very suggestive symptom of pituitary pressure, and, in the absence of any indication to the contrary, it should, at any rate provisionally, generally be regarded in this light.

This sequence of events as regards loss of vision is, however, by no means constant, for it frequently happens that both visual fields contract without any preliminary discrimination for the temporal half being observed, though it is not often that both eyes are equally affected until, at any rate, the later stages in the disease have been reached. In some instances homonymous hemianopia may be the first change, and in others a central scotoma expanding into the temporal field may be a notable feature of the failing sight. Failure of vision for colours has been observed to occur before that for form, and according to Cushing this order of events is the rule.

Herbert Fisher considers that the visual symptoms in many cases are best explained by the occurrence of traction on the visual pathways, as the tumour extends upwards behind the chiasma and between the optic tracts in the interpeduncular space. He thinks that a tumour which is fairly symmetrical in outline might stretch the decussating fibres in the chiasma without dragging on the direct fibres, and so cause a bitemporal hemianopia. On the same hypothesis can be explained the cases of expanding scotoma, and hemiachromatopsia can also be more easily accounted for than on the assumption of the direct-pressure causation. There is evidence, moreover, to show that the loss of function of vision is often in advance of the apparent structural changes; and from the recovery of vision which has resulted from relief of pressure, Cushing concludes that "it is safe to say that the amblyopia associated with a primary atrophy more often represents a physiological block to light impulses than an actual destruction of the nerves."

Headache in pituitary tumours is due to rise of intracranial pressure. It is, however, important to note that the headache is not necessarily relieved by the ordinary decompression operation, because the gland is closed in by dura mater in such a way as to form a local sac, which must itself be tapped if adequate relief is to be obtained. Mucous discharge into the pharynx may occur as the result of changes in the neighbouring sphenoidal sinuses. Enlargement and alteration in shape of the sella turcica often occurs, and when, as frequently happens, it can be detected by means of the X-rays, it becomes a fact of great diagnostic importance.

The changes due to alteration in general metabolism show themselves chiefly in overgrowth of the skeletal tissues.

In the head (Fig. 97), enlargement of the upper and lower jaws is among the most prominent characteristics, and the face assumes a heavy and elongated aspect. At the same time the features become broadened and coarsened owing to changes in the soft tissues and cartilages. The bony sinuses become dilated, thus causing undue prominence of the cheeks and of the orbits, and giving a peculiar, massive expression to the countenance. The nose becomes thickened, chiefly due to the increase in thickness of its cartilage, and the cartilages of the ear are often subject to changes of a similar character. A change in the bones which almost invariably takes place sooner or later is curvature of the spine (Fig. 98), and indeed this symptom ranks among those which are most typical of the disease. The vertebræ enlarge and form an antero-posterior as well as a lateral curve. Of the two, the antero-posterior curve is usually the greater, and causes the rounded shoulders and the hanging head—which can only with difficulty be held up straight. The bones of the thorax are also altered. The hands and feet are early and greatly enlarged, and it was from this fact that Marie called the condition acromegaly.

The changes are not by any means limited to the bones; the subcutaneous tissue becomes infiltrated in a manner somewhat similar to the infiltration which occurs in myxœdema, with the result that the parts become swollen and puffy. In the face this swelling and puffiness add to the coarsening of the features, which

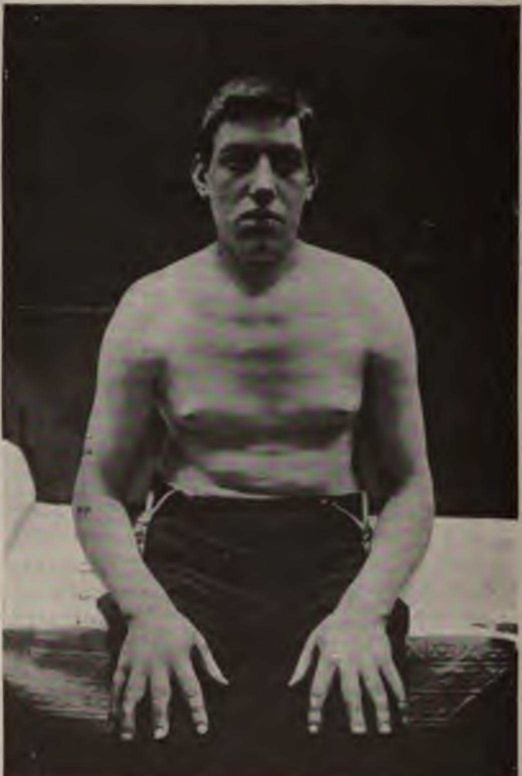

Fig. 97.—A typical case of acromegaly.

are already broadened by the changes in the bones and cartilages. This puffiness is very noticeable in the lips, and more especially in the lower lip, which assumes a thickened, pouting, overhanging appearance. The tongue is increased in size and becomes too large to fit comfortably in the mouth. The hands are broadened, with the ends of the fingers blunted, and have been compared in their shape to a spade or a battledore. The feet undergo changes of a similar nature.

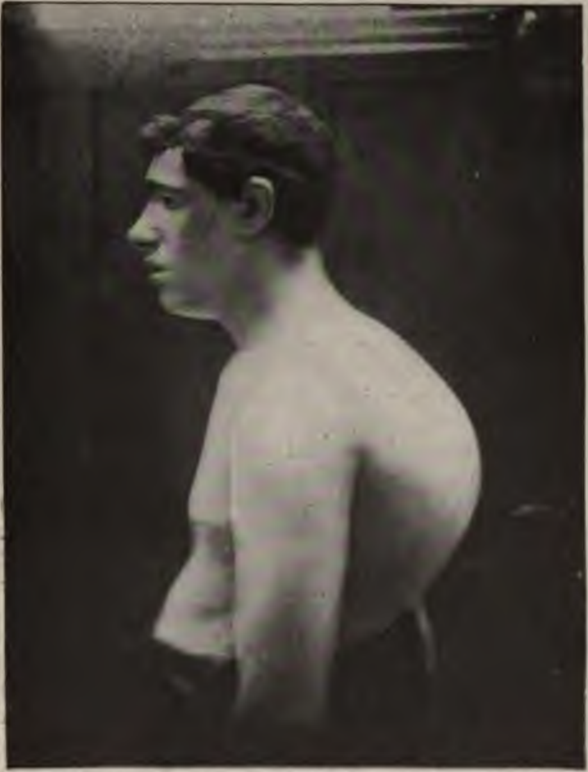

Fig. 98.—Side view of same patient as in Fig. 97, showing the curvature of the spine.

Alterations in the size of the thyroid have frequently been noted, and as a general rule the change is in the direction of diminution. An area of dullness over the upper portion of the sternum has sometimes been observed, and has been attributed to an increase in the size of the thymus gland, while the occasional presence of sugar in the urine is suggestive of a disturbance of the secreting properties of the pancreas.

Transient symptoms of acromegaly have been occasionally observed to occur during pregnancy, due presumably to changes in the pituitary gland analogous to those which sometimes occur in the thyroid during this state.

Treatment.—Medicinal treatment is practically limited to the use of preparations of the thyroid or pituitary gland.

The benefit obtained from pituitary extracts is variable, but they are indicated when there is reason to believe the secretion of the gland is diminished.

Thyroid preparations undoubtedly do good in some cases, both by improving the sight and by checking the progress of the more general symptoms of the disease.

The explanation of the beneficial action of thyroid is to be found in the close relationship that exists between the functions of this gland and those of the pituitary body.

It has been shown experimentally that the removal of the thyroid in animals may be followed by an enlargement of the pituitary gland and a modification of its secretion, and a similar tendency to enlargement has been observed clinically in cretinism and in other instances of thyroid deficiency. If, then, insufficiency of thyroid secretion is accompanied by some compensatory process on the part of the pituitary body, as appears to be the case, it seems permissible to assume that, in certain circumstances, administration of thyroid may diminish the pituitary functions and lead to a decrease in the size of the gland.

However this may be, there remains the clinical fact that patients do frequently show some improvement after taking thyroid, especially with regard to their sight, and in the present state of knowledge this method is probably as rational a mode of procedure as can be adopted. Schweinitz thinks the value of the thyroid is enhanced when given in association with mercurial inunctions.

Exposure to X-rays has been recommended as a means of causing pituitary tumours to shrink, and beneficial results are said to have occurred. Presumably the action would be similar to that which the rays exert on enlarged thyroid glands.

When the local pressure symptoms are great, the question of the possibility of obtaining relief from a surgical operation has to be considered. The possibilities in this respect comprise: (1) relief of symptoms by a local decompressive operation to relieve the tension in the sella turcica; (2) drainage of a cyst should one be found to be present; (3) partial removal of an adenomatous or malignant growth.

Any operation on the pituitary gland is necessarily one of difficulty and severity, and for the various routes and methods of procedure the reader is referred to Cushing's* other special surgical works on the subject.

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* "The Pituitary Body and its Disorders."

CHAPTER XXXIX

PROGRESSIVE LENTICULAR DEGENERATION

An uncommon disease under the title of "progressive lenticular degeneration," which occurs in young people and tends to run in families, has been described by Kinnier Wilson.*

Symptoms.—Among the principal symptoms are tremors, muscular rigidity, difficulty and pain in swallowing, spasmodic contractions and contractures. The patients are emotional, and mental symptoms may be present; the reflexes in uncomplicated cases are normal, but they may be modified if the pyramidal tracts are secondarily affected.

Morbid anatomy.—Bilateral degeneration of the lenticular nucleus, especially of that part known as the putamen, is the principal lesion met with in the brain, while, of the other organs, cirrhosis of the liver which does not cause any symptoms during life, and is not of syphilitic or alcoholic type, is constantly present, and is regarded by Wilson as being the probable primary factor of the disease.

Prognosis and treatment.—The disease is progressive, but uncertain in its duration. An average length of time for chronic cases is given as four years, but in some instances a fatal termination has occurred in a few months. The disease does not react to any known form of treatment.

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* *Brain*, vol. xxxiv., and Pt. iv., 1912.

CHAPTER XL

CEREBRAL HÆMORRHAGE; CEREBRAL THROMBOSIS; CEREBRAL EMBOLISM

Arterial supply of the brain.—The blood supply of the brain is derived from the internal carotid and vertebral arteries. On reaching the base of the brain each internal carotid divides into two branches, which are known as the anterior and middle cerebral arteries. The anterior cerebral arteries run forward and, passing over the genu of the corpus callosum, continue their course in the median fissure to supply the anterior part of the frontal lobes and the inner surface of the hemispheres as far back as the parieto-occipital fissure. The two anterior cerebral arteries are united, soon after their origin at the base of the brain, by a branch that is known as the anterior communicating artery. The other branch of the internal carotid, namely, the middle cerebral artery, runs along the Sylvian fissure until it reaches the island of Reil, where it divides into terminal branches which supply the outer surface of the hemisphere. The middle cerebral arteries are responsible for the blood supply of the whole of the outer surface of the cerebral cortex, with the exception of two narrow strips, one on the anterior border of the frontal lobe and another at the lower border of the temporo-sphenoidal and occipital lobes. (*See* Fig. 99.)

As the middle cerebral artery runs across the base of the brain it gives off the lenticulo-optic and lenticulo-striate branches to the corpus striatum, which branches, owing to the poorness of their anastomosis, are known as "end arteries." The lenticulo-striate branch runs along the outer side of the lenticular nucleus and supplies the region of the internal capsule. It is notorious for its liability to rupture, for which reason it has been called the *artery of cerebral hæmorrhage.*

Another result of the poorness of anastomosis of the central arteries is that any embolic or thrombotic affection of them is likely to be followed by permanent softening of that part of the brain which they supply.

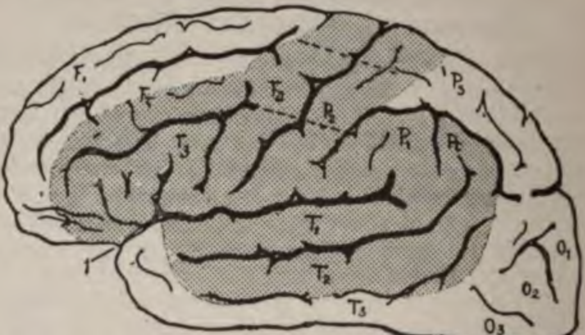

Fig. 99.—Area of softening of the cortex due to obliteration of the Sylvian artery at its commencement. (*Pierre Marie.*)

The two vertebral arteries run along the surface of the medulla, and on reaching the pons unite to form the basilar artery; but each vertebral just before uniting with its fellow of the opposite side gives off an important branch, the posterior inferior cerebellar artery, which runs obliquely backwards round the medulla, to which it gives branches, and then passes on to form an important source of blood supply of the cerebellum. The basilar artery supplies branches to the pons, and after giving off the anterior inferior and superior cerebellar arteries it breaks up to form the two posterior cerebral arteries. The posterior cerebral arteries wind round the crus cerebri, and supply the lower parts of the temporal and the occipital lobes. Each artery also communicates with the internal carotid by the posterior communicating artery, the circle of Willis being thus established at the base of the brain.

CEREBRAL HÆMORRHAGE

Etiology.—Arterial degeneration and an accompanying rise of blood-pressure are the main factors concerned in the production of cerebral hæmorrhage.

These two conditions are most often associated with *chronic interstitial nephritis,* and it is during the course of this disease that a large number of cases of hæmorrhage occur.

Naturally, the causes which lead to the final rupture of a vessel are often complex and difficult to analyse, since they include all those which tend to produce arterial degeneration: some of the principal of these are *alcoholic excess, chronic lead-poisoning, syphilis,* and *senile degeneration.*

It is particularly important to remember the tendency shown by the blood-pressure to rise during the later years of middle life, even when there are present no recognizable signs of renal disease; and no doubt, as Sir Clifford Allbutt has strongly insisted, a more general and timely recognition of this fact, combined with suitable treatment, would lead to many attacks of apoplexy being averted or postponed.

The obliterative endarteritis from syphilis more often leads to thrombosis than to hæmorrhage, as also does the degeneration which accompanies very old age.

The arteries of men break more often than those of women, and the period in which most cases occur is from 45 to 65 years of age. Sudden exertion may precipitate the attack, but in a considerable number of instances the artery breaks while the patient is asleep. Hæmorrhage sometimes takes place into a tumour of the brain, and it may also be associated with changes in the blood, as in leucocythæmia, pernicious anæmia, purpura, septicæmia, and scurvy, but the symptoms produced in these latter cases are seldom those of typical apoplexy.

Pathology.—The arteries in a case of cerebral hæmorrhage generally present the usual appearances of an advanced stage of arterio-sclerosis. Their walls are rigid and brittle, and small aneurysmal dilatations, the rupture of one of which is the cause of the hæmorrhage, can frequently be seen with the naked eye or a magnifying glass.

The appearance of the blood differs according to the length of time which has elapsed since the hæmorrhage occurred. At first there are all the appearances of freshly extravasated blood, but, after a clot has formed, a gradual shrinkage takes place, and the last appearances may be those of a scar or a cyst with discoloured walls containing some clear or brownish fluid.

Symptoms.—Symptoms which depend on degenerated vessels and high blood-pressure, such as headache, giddiness, tingling and numbness of a limb, are often complained of by people whose arteries are likely to rupture, and sometimes more distinct warnings, such as a ptosis, squint, or aphasia of passing duration, are met with. Many such signs are probably due to minute hæmorrhages, the tendency to which may often be seen by looking at the retina, but it is also possible that they may be produced by arterial spasm, though the occurrence of spasm of cerebral arteries is a matter about which there is a good deal of difference of opinion.

A hæmorrhage causes symptoms (1) by the rise of intracranial pressure which it produces, and (2) by destroying the function of the nerve fibres in the locality in which it occurs.

When a hæmorrhage is quite small there may be symptoms of local injury without any disturbance of consciousness, and it is probably in this way that the temporary ptosis and squints sometimes preceding a more severe attack are produced.

On the other hand, consciousness may be considerably disturbed without any permanent local effect.

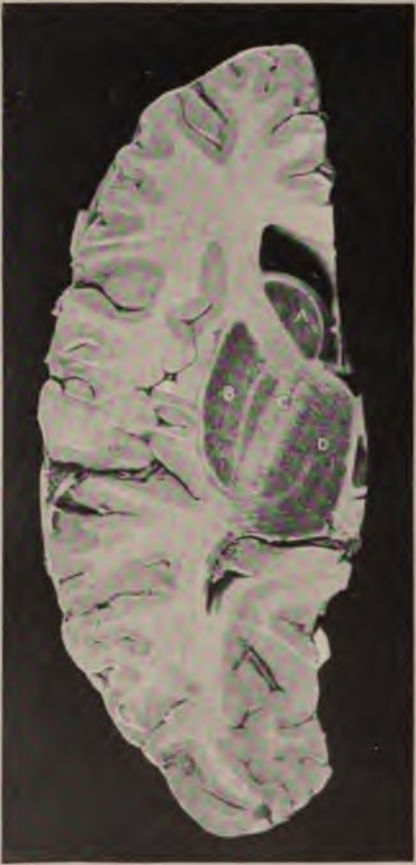

PLATE 21.—Section of brain showing the anatomy of the internal capsule.

A, Caudate nucleus; B, lenticular nucleus; C, internal capsule; D, optic thalamus.

Classification of symptoms.—It is convenient to classify the symptoms into:

(1) *General*—common to all hæmorrhages of a certain size, irrespective of their situation.
(2) *Local*—depending on the position of the hæmorrhage.

(1) The chief **general symptoms** are: *loss of consciousness*, varying in degree from confusion to coma; *stertorous breathing*, which may be of the Cheyne-Stokes type; *slow full pulse*, *subnormal temperature*, and loss of power over the sphincters (this last depends on the depth of unconsciousness).

(2) The **local symptoms** necessarily vary with the position of the hæmorrhage, which may be anywhere in the brain, but the cerebral vessels which most often break are the branches of the *lenticulo-striate* artery, which come off directly from the middle cerebral and pierce the base of the brain to supply the neighbourhood of the lenticular nucleus. These vessels are in close proximity to the motor fibres as they pass through the internal capsule, and an effusion of blood of any size in this neighbourhood nearly always causes loss of power on the opposite side of the body. (Plate 21.)

The hæmorrhage is usually large enough also to cause loss of consciousness, and hence, in the greater number of cases, the symptoms bear a close resemblance to one another.

As a *typical example*, it is therefore convenient to discuss the results of *hæmorrhage* and *hemiplegia* resulting from rupture of the *lenticulo-striate artery*.

When the attack takes place in the daytime the patient feels giddy and, after perhaps reeling for a few steps, sinks in a dazed manner to the ground or on to a chair, and quickly loses consciousness. It is usually after this condition has existed for some little time that he is seen by the physician; the patient is then completely unconscious, breathing stertorously, and has a full, slow, bounding pulse and a turgid countenance. Perhaps the head and eyes are persistently turned to one or other side.

Diagnosis.—An attempt at this stage must first be made to determine the *cause* of the *coma*, but for a time it may be impossible to arrive at any definite conclusion.

The chief conditions besides cerebral hæmorrhage that suggest themselves in such a case are *uræmia diabetes*, *alcohol*, *opium* or other *narcotic*, *postepileptic coma*, and *concussion*.

In *uræmia* the attacks may for a time very closely resemble those of cerebral hæmorrhage, especially if, as sometimes happens, the power of movement on the two sides of the body is unequal. Moreover, cerebral hæmorrhage is likely to occur in the very people who are also liable to uræmia. In some cases, twitchings and convulsions, together with the history of the previous symptoms, afford some assistance, but in others the diagnosis must for a while remain in doubt.

Coma due to *alcohol* often gives rise to difficulty, and the danger lies in pronouncing a man to be drunk when he has been the subject of a hæmorrhage while drinking. The evidence of drink does not exclude the possibility of hæmorrhage, and, when there is the least doubt, the only safe course is to keep the patient under careful observation for a few hours.

The coma of *diabetes* generally comes on rather more gradually than that of hæmorrhage. The dyspnœa is that of the "air hunger" type, and lacks the deep stertor that accompanies hæmorrhage, nor is there any sign of unilateral paralysis. If a specimen of urine can be obtained the presence of sugar will make the diagnosis clear.

The signs of *opium* poisoning closely resemble, in the contracted pupils and deep coma, those of hæmorrhage into the pons. Fortunately there are generally some indications in the history which lead to the suspicion of poisoning.

The coma which follows an *epileptic fit* is sometimes very deep, but there is generally an account of the convulsion even if the history of a previous liability to fits be not obtainable.

In *concussion*, the difficulty of deciding whether there is a meningeal hæmorrhage may arise, for it very rarely happens that an intracerebral artery ruptures from the direct effects of a blow. When a meningeal artery bleeds, the signs are those of an increasing pressure on the brain, possibly accompanied by convulsions.

In the course of the examination of a case of supposed cerebral hæmorrhage, careful observation often detects signs of one-sided weakness. One cheek puffs out loosely, and the arm and leg on the same side when raised fall in a heavy, dead fashion, which, in spite of the loss of consciousness, obviously differs from that of the other side and justifies the conclusion that a one-sided cerebral lesion is the cause of the trouble.

The ground is cleared considerably when this conclusion can be arrived at, since a one-sided lesion coming on suddenly in a man who has up to that time been in ordinary health almost certainly indicates a vascular lesion, and so the issue is narrowed to *hæmorrhage, thrombosis*, or *embolism*.

Embolism must be excluded by the absence of a cause, such as valvular disease of the heart, and moreover the deep loss of consciousness would at once weigh heavily against it; for although blocking of the middle cerebral artery or one of its branches will cause a good deal of mental confusion, especially if complicated with aphasia, there is nothing in its nature to produce the deep coma that accompanies the rapid rise of intracranial pressure consequent on the effusion of a quantity of blood.

In the diagnosis between *thrombosis* and *hæmorrhage*, often a most difficult matter, the main points to be considered are—

1. *Loss of consciousness.*—The existence of extensive paralysis without any definite loss of the senses is highly suggestive of thrombosis, for a sudden effusion which is large enough to cause hemiplegia is most likely also to raise the intracranial pressure sufficiently to cause loss of consciousness. Of course, an exception to this

general rule may occur when a small hæmorrhage takes place somewhere exactly in the course of the motor fibres.

2. *Onset of paralysis.*—In the case of hæmorrhage the onset of paralysis is generally sudden and does not show signs of extension, for the damage is usually all done in a very few minutes. On the other hand, a paralysis which progresses in depth and distribution over a period of some hours, especially if there is no loss of consciousness, is very characteristic of thrombosis in which the clot is extending from the original thrombus into neighbouring vessels. Before the onset of paralysis there may have been numbness, tingling, or a passing weakness in a limb, signifying that the blood supply was very deficient before the occlusion finally occurred.

3. *The vascular system.*—The association of an hypertrophied heart with degenerated arteries is in favour of hæmorrhage. High arterial tension also disposes to hæmorrhage, and further evidence may be obtained from signs of the existence of chronic interstitial nephritis.

4. *Age.*—In the very old, thrombosis is more likely to occur than hæmorrhage. In the young and in early middle life, say under 40, cerebral hæmorrhage is rare, while thrombosis is comparatively frequent from syphilitic endarteritis.

Treatment during the early stage.—As soon as the diagnosis of cerebral hæmorrhage has been made it is necessary to proceed at once to treat the patient.

In rare instances, where the patient is seen at the moment the bleeding is taking place, prompt venesection and pressure on the carotid arteries might be of use; but, as a general rule, the time for these measures has gone before any opportunity of treating the patient arises. The best method then of trying to prevent further oozing is to keep the patient at absolute rest, and to calm down the circulation by relaxation of the bowels to a moderate degree, such as can be obtained by calomel (gr. ii to v) or croton oil (♏ ½ to i), followed if necessary by an enema.

The patient's head, upon which an ice-bag may be placed, should be raised and turned rather to one side, in which position the stertor is often diminished. For the first few hours no food is necessary; after that a little milk may be given. When necessary the urine should be drawn off, great care being taken to keep the skin clean and dry.

Prognosis.—The next step which it is usually necessary to take is to give an opinion as to the danger to life, and in this connection the chief points to observe are—

(1) The *depth of the coma.*
(2) The *course of the temperature.*
(3) Whether *conjugate deviation* of the head and eyes is present.

A deep *loss of consciousness* suggests that either the hæmorrhage is large or situated in some specially important part of the brain, as, for instance, the neighbourhood of the pons or medulla; in both cases life is in danger. Of particularly evil significance is a coma which deepens progressively, for it usually indicates that the bleeding is still going on, or that the blood is finding its way into the lateral ventricles. Generally speaking, if there are no distinct signs of returning consciousness in twenty-four hours the outlook is bad. On the other hand, slight loss of consciousness and signs that the coma is beginning to pass off are both satisfactory indications.

Immediately after the hæmorrhage has taken place there is a sudden fall in the *temperature* to two or three degrees below normal, which is followed by a rise above normal when the preliminary shock has passed off. If this rise keeps within the limits of one to two degrees above normal the outlook is favourable; but if the temperature is raised above this for any length of time, and shows signs of being raised still more, it is a sign that the patient will probably die.

Conjugate deviation of the eyes and head, especially if it persists, is a sign of bad omen.

Other symptoms, such as the state of the pupils and the presence of Cheyne-Stokes breathing, may also help in making a prognosis.

Signs of hemiplegia.—With return of consciousness the local effects of the lesion become more apparent, and when the hæmorrhage is in the region of the internal capsule there are often sensory as well as motor symptoms.

1. **Motor symptoms.**—There will most likely be loss of power of the upper-neurone type for voluntary movements in the face, arm, and leg on the opposite side to the lesion.

Face.—In examining the face there are three sets of movements to be considered, viz. :

(1) *Unilateral voluntary movements*, i.e. voluntary movements that are habitually made on one side of the face, e.g. drawing up the corner of the mouth.

(2) *Bilateral voluntary movements*, i.e. voluntary movements in which both sides of the face are habitually put into action at the same time, e.g. closing the eyes and wrinkling the brow.

(3) *Emotional movements*, e.g. those of laughing and crying, into which volition only enters to a slight degree.

In hemiplegia these three sets of movements are affected in very different degrees.

The unilateral voluntary movements are the most highly specialized, and the path by which impulses for their production can pass from the motor cortex to the muscles is confined to the direct course of the motor fibres. Interruption of these fibres leaves no alternative route, hence these particular movements are lost.

Bilateral voluntary movements are less specialized, and there is still some communication possible between the nuclei of their nerves on the two sides. When, therefore, the path from the cortex is interrupted on one side, it is still possible to obtain impulses through the communication with the nucleus on the other side, and so in hemiplegia the voluntary bilateral movements, though often temporarily weakened, do not suffer to such an extent as the unilateral ones. The patient is thus enabled to wrinkle the brow, frown, and close the eyes, even though he cannot move the corner of his mouth.

Reference has already been made to the lateral deviation of the head and eyes which may take place in severe cases (p. 395).

Emotional movements are still less diminished, and the corner of the mouth often moves freely during laughing or crying when it cannot be moved by the will.

The *tongue*, when put out, usually deviates towards the paralysed side, and on phonation the two halves of the *soft palate* are unequally drawn up.

The *arm* is generally deeply paralysed, the finer movements of the fingers suffering more than the coarser ones of the elbow and shoulder.

The *leg* is usually less deeply paralysed than the arm, and is nearly always the first to show signs of returning power.

In hemiplegia of organic origin, Babinski has shown that if the patient lies on his back with his legs widely separated and attempts to assume a sitting position, the paralysed leg is, in the attempt, lifted higher from the floor than the sound limb—a phenomenon which forms one of the distinguishing features between organic and functional disease.

The movements of the *trunk* are for the most part bilateral, and, although slight differences can be detected between the respiratory movements of the two sides, they are not usually of special clinical importance.

2. **The reflexes.**—The tendon reflexes on the paralysed side are exaggerated, as is the rule in upper neurone lesions, and after a time there is sometimes an increase in those of the opposite side also. In the leg the knee-jerk and tendo-Achillis jerk are very active, and ankle-clonus can usually be obtained. The plantar reflex

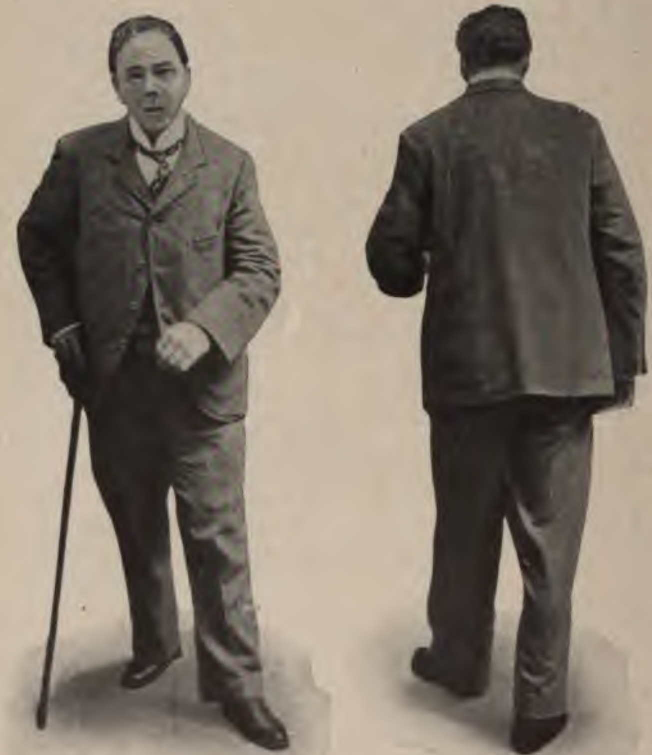

PLATE 22.—Characteristic gait in hemiplegia.
The rigid and weakened left leg is stiffly circumducted from the hip.
Note also the characteristic position of the paralysed arm.

walking the whole limb is stiffly swung round in the arc of a circle from the hip, i.e. circumducted (Plate 22).

The hypotheses which have been brought forward to account for this rigidity and the methods necessary to adopt in order to avert it are discussed at p. 39.

Involuntary movements.—Hemiplegic conditions are sometimes accompanied by involuntary movements known as post-hemiplegic chorea and athetosis. Such movements appear more prone to occur in lesions of the thalamus and its neighbourhood, and are apparently due to release from control with consequent overactivity of subsidiary motor systems (*see* p. 10). These movements are very apt to arise in the hemiplegias and diplegias of childhood, but their onset may be postponed for a considerable time after the date of the original lesion.

Post-hemiplegic chorea is characterized by tremors or irregular movements in the arm or leg of the paralysed side.

Athetosis is characterized by involuntary movements which are generally most pronounced in the hand or foot, but occasionally the whole limb may take part, as also may the face.

In the hand, the fingers move in a slow manner, and are alternately flexed and hyperextended. All the fingers do not move equally, but one or more at a time are hyperextended in a fashion which it is not possible to imitate. In some instances the movements go on almost continually, but in others they are often started by an attempt to move the limb voluntarily. In severe cases the arm may be thrown about in such a fashion that it becomes necessary to bind it to the patient's side.

Associated movements are also met with in hemiplegics. The paralysed arm is often strongly drawn across the chest when the patient yawns or breathes deeply, and the movement of one leg may be accompanied by some movement of the other.

When all signs of improvement in the power of the limbs cease, and rigidity makes its appearance, there is not much hope of any further improvement so far as recovery of the functions of the injured neurones is concerned. Treatment must, however, on no account be abandoned, for by improving the function of fibres that remain, and by warding off rigidity and contractures, much can be done to render the limb a useful one. If the methods described at p. 41 are persevered with, marked benefit can often be obtained; and it must always be remembered that the difference between a helpless limb and one that can be used, however slightly, is of the greatest importance to the patient.

Pain.—In some cases pain in the paralysed limbs is a very distressing symptom. It is very apt to occur in association with lesions of the thalamus. As explained on p. 61, it may be due to over-activity of the thalamus rather than to persistent irritation of sensory fibres, which is an explanation frequently suggested.

Hæmorrhage into the pons and crus cerebri.—Hæmorrhage into the pons is usually fatal within a few hours.

Deep coma, contracted pupils, and hyperpyrexia are among the characteristic signs. Convulsions may also occur. There is often paralysis on both sides of the body, since a hæmorrhage of any size will injure both sets of motor fibres.

If, however, the hæmorrhage is small and situated at the upper part of the pons or in the crus cerebri, it is possible to get "crossed paralysis," i.e. of the face on one side and of the arm and leg on the other, owing to the fibres of this nerve crossing at a higher level than those for the arm and leg, which do not decussate till they reach the medulla.

The fibres to the ocular muscles may also be affected in lesions of the pons.

The association of coma and contracted pupils makes it necessary to bear in mind the possibility of opium-poisoning when the paralysis is not obvious and no history of the case is available.

Summary of the causes of hemiplegia.—The symptoms of hemiplegia following cerebral hæmorrhage as described in the previous paragraphs will serve as a general example, but it is also necessary to remember the other principal causes of this condition, since the patient may be seen for the first time long after the occurrence of the original lesion.

The causes of hemiplegia may be classified somewhat as follows:

1. *Vascular lesions* ...	Hæmorrhage.	Meningeal. Intracerebral.
	Thrombosis.	
	Embolism.	
2. *Inflammations* ...	Meningitis.	Localized, or especially intense over the motor areas.
	Acute encephalitis.	
3. *Tumours*	Pressing on the motor tracts.	
4. *Degenerations* ...	Degenerative processes of the brain, e.g. sclerosis.	
5. *Toxæmias*	In uræmia, the effects of the poison are sometimes especially visited on the motor areas, and a hemiplegia which is often difficult to distinguish from that of cerebral hæmorrhage may occur.	
6. *Exhaustion following convulsions*	Temporary hemiplegia, presumably due to exhaustion, may follow a severe epileptic fit.	
7. *Functional hemiplegia*	Hemiplegia is a not uncommon manifestation of hysteria.	

CEREBRAL THROMBOSIS

Etiology.—The chief conditions which lead to thrombosis are *atheroma* of the vessels and a *feeble circulation;* hence, thrombosis is more likely to occur in advanced years, when the vessel walls are thickened and the circulation slow. On the other hand, in the later part of middle life, when the arteries are apt to be weakened, while the force of the heart still remains strong, there is a greater liability to hæmorrhage.

Among the principal causes of arterial degeneration which may eventually lead to thrombosis are syphilis, alcohol, and Bright's disease, and of these syphilis is the most important. The atheroma of Bright's disease is more likely to be associated with hæmorrhage than with thrombosis.

Syphilitic endarteritis is the commonest cause of the cases of cerebral thrombosis that are met with before the occurrence of the degenerative changes of senility. The proliferation of the tissues of the arterial coats gradually narrows the lumen of the vessel until finally the flow of blood is checked and the parts beyond are deprived of their nutrition.

In some instances the cause of the thrombosis depends upon changes in the *composition of the blood.* This is the case in thrombosis associated with severe anæmias and with some varieties of septicæmia. Allusion has already been made to infective processes as a cause of thrombosis in infantile paralysis and the cerebral palsies of children.

Symptoms.—The symptoms vary according to the mode of onset and the locality of the vessel that is blocked.

1. **Gradual onset.**—When the lumen of the vessels becomes narrowed gradually, the first symptoms are likely to be those due to diminished blood supply to certain parts of the brain, i.e. to cerebral anæmia; the function of the part is then apt to be impaired without being altogether lost, and may perhaps again be restored as some variation takes place in the state of the local circulation. Thus, before there are signs of complete thrombosis, there may be warnings in the guise of temporary failure of power in a limb, an ocular muscle, or an eyelid, or there may perhaps be some loss of memory, with a temporary condition of partial aphasia; while in other cases the patients complain of tingling, numbness, and giddiness. One day matters may go farther: a clot forms, and the area supplied by the vessel is cut off from the circulation. With a gradual onset the

general symptoms of cerebral disturbance are not likely to be great, and in many instances consciousness is never lost.

2. **Sudden onset.**—A more sudden onset is the characteristic of other cases where the function of the part has not been perceptibly impaired before the vessel becomes completely blocked. Paralysis of one or more limbs sets in suddenly, and the general cerebral disturbance is then often sufficiently great to cause loss of consciousness, though the coma is seldom so deep as that of a hæmorrhage causing symptoms of a corresponding extent. Some idea of the effects of thrombosis of different arteries and their branches can be gauged from the diagram which shows the area of softening after blocking of the middle cerebral (Fig. 99, p. 388).

If, as frequently happens, a branch of the middle cerebral artery to the motor cortex is blocked, there will be loss of power corresponding to the extent of brain affected. If, for example, the branch that is blocked happens to be that supplying the motor area of the cortex in which movements of the arm are represented, the limb will be paralysed, and will also very likely be subject to attacks of localized convulsions owing to the irritation of the cerebral cortex.

When a clot has formed it frequently extends into other branches, and thus a monoplegia may in a few hours be succeeded by a hemiplegia.

If, however, the main trunk of the middle cerebral artery is blocked, a complete hemiplegia results from the first, and to this, if the lesion is on the left side of the brain, is added aphasia.

An equally severe hemiplegia, with perhaps hemianæsthesia and hemianopia, may also result from thrombosis of the vessels supplying the motor fibres of the internal capsule, but speech is not in these circumstances often permanently lost.

Where the tendency to thrombosis is widespread, loss of memory and general mental failure may be the most prominent features of the case.

Thrombosis of the posterior inferior cerebellar artery also causes characteristic symptoms, which usually include some degree of incoördination of the limbs on the same side as the lesion, nystagmus, strabismus, and disturbances of sensation over the half of the face on the side of the lesion, with diminished sensation to pain and temperature, and impaired position-sense on the limbs and trunk on the side opposite the lesion.

Pathology.—An area of the brain that is deprived of its blood supply becomes softened and necrosed. Sometimes it remains pale, but often there is some transudation of blood, as in the case of infarcts of other organs; and pale, red, and yellow softening are spoken of, according to the coloration produced by the blood. In late stages a cyst or a scar may represent the area of disease.

Prognosis.—In giving a prognosis of the results of thrombosis, it is necessary to try to form a mental picture of what is taking place at the seat of the disease. The blockage of an artery causes *total anæmia* of any portion of brain which happens to be entirely dependent on it for its nutrition, and *partial anæmia* of the parts around, which can still obtain some nutrition from collateral sources.

Hence the first effect is destruction of the function of the nerve cells in the area which the vessel wholly supplies, and a lowering of the function of those parts on the border of that area which obtain some of their blood supply from elsewhere. Unless the circulation to the part which is wholly dependent on the thrombosed artery is restored quickly, the nervous tissue will disintegrate and be incapable of repair. On the other hand, the function of the parts in the immediate vicinity, depressed as the first effect of the arterial blockage, improves to a considerable extent as the collateral circulation becomes better established. So that when, after a few days, there is no return of power, it may be fairly assumed that permanent damage has been done, though a certain degree of recovery may still take place as the function of the surrounding part improves.

Thrombosis of the vessels supplying the region of the internal capsule is especially apt to be followed by permanent softening, for the collateral circulation of that part of the brain is very poor and can only be very feebly maintained from the terminal branches of the cortical arteries.

The outlook is bad in those cases in which gradual failure of the mind signifies a widespread softening.

Diagnosis.—The diagnosis from cerebral hæmorrhage has been considered earlier in the present chapter (p. 393).

Treatment.—If the danger is recognized in time, as it may be when the circulation is weak and the arteries are obviously thickened, much can be done to avert thrombosis by increasing the rate of the blood-flow and by modifying the nutrition of the vessel walls; but, once the clot has formed, no method of treatment will ensure its dispersal, nor can any drug restore the vitality of a patch of softened brain.

In those who have syphilitic endarteritis, a course of mercury and iodides would doubtless often prevent catastrophe; but too often they seek no treatment till the event has taken place, and the most that can usually then be done is to try to prevent a recurrence.

In every case of recent thrombosis due to syphilitic disease of the arteries it is a good plan to prescribe antisyphilitic treatment, and afterwards, with a view of anticipating recurrence, to give regular courses of iodide at stated intervals, to which, if it seems desirable at any time, injections of salvarsan or a course of mercury may be added.

During the attack the patient should be kept quiet and fed lightly. Care should be taken not to purge severely and not to adopt any other measures that are likely to depress the force of the circulation, which may often be improved with advantage by a little digitalis or other suitable cardiac stimulant.

CEREBRAL EMBOLISM

Etiology.—Embolism is nearly always associated with endocarditis of the aortic or mitral valve. Occasionally it arises from detached portions of clot from an aneurysmal sac.

In chronic endocarditis, mitral stenosis is the form most likely to cause embolism, but in the acute infective forms a fragment may be dislodged from either the aortic or mitral cusps.

Emboli are on the whole more likely to pass up the left carotid artery, and so it is more common to get a lesion of the left than the right side of the brain.

Symptoms.—The onset is quite sudden and is accompanied by symptoms which vary according to the position in which the clot lodges. If, as often happens, the main branch of the middle cerebral artery on the left side is blocked, hemiplegia and aphasia immediately result. In such a case the senses are often lost, but there are seldom the depth of coma, stertorous breathing, and disturbance of temperature that are met with in cerebral hæmorrhage.

Softening of the cortex will probably be accompanied by convulsions, but these will be absent when the obstruction occurs in the central branches of the artery after they have left the main stem, though the hemiplegia may be complete from softening of the fibres of the internal capsule.

When only a small branch of the middle cerebral is blocked the symptoms are, of course, less severe. Embolism of the posterior cerebral artery causes hemianopia, and (according to Marie) visual aphasia also.

If thrombosis starts from the original clot and extends along the vessels, the sudden symptoms at the beginning are followed by others of more deliberate progress.

Prognosis.—What has been said regarding the prognosis in cases of cerebral thrombosis may be in a large measure applied to cerebral embolism. In both, the restoration of cerebral function depends upon the possibility of the rapid re-establishment of the circulation, for when softening has once occurred in a part, its function cannot be regained, though it may perhaps be partially compensated by the action of other parts.

Diagnosis.—The diagnosis between hæmorrhage, thrombosis, and embolism is discussed at p. 393.

Treatment.—The clot cannot be modified in any way. The after-treatment is that which is detailed for hemiplegia, and consists in massage and correction of faulty positions of the paralysed limbs, while the patients must be encouraged to develop any return of voluntary power that may occur.

CHAPTER XLI

THROMBOSIS OF CEREBRAL SINUSES

THROMBOSIS of the cerebral sinuses may arise as the result of general diseases, e.g. severe anæmia, wasting and diarrhœa (especially in infants), and typhoid fever, or it may take place after a local injury. In these cases the thrombosis is often called "simple," as opposed to the infective variety, which is the more important, and which arises as the result of some near or distant septic condition. Suppuration of the middle ear or of the nasal sinuses is the most important local cause of infection.

Thrombosis of the lateral sinus.—This is especially apt to be associated with chronic suppuration of the middle ear, and it may coexist with cerebral abscess.

The chief **local signs** are pain, œdema, and distension of veins over the mastoid region, with a sense of hardness over the course of the internal jugular vein, down which the clot frequently extends. The **general symptoms** are mainly those of septic infection, viz. high temperature, rapid pulse, and delirium.

This condition gives cause for grave anxiety. The best mode of treatment is to open up the sinus and clear out the infective clot, and at the same time to deal with an abscess should it be present.

Thrombosis of the cavernous sinus.—Sir StClair Thomson has shown * that thrombosis of the cavernous sinuses is especially liable to follow chronic suppuration of the nasal cavities.

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* *Trans. of Med. Soc. of London*, 1906.

The main **general symptoms** which indicate the onset are, according to Sir StClair Thomson, acute headache, delirium, and drowsiness, with sickness, high temperature, and rapid pulse. The headache is generally severe, and the pain may be referred to the side of the head, to the back of the ear, or to the frontal region. With these there are generally some **ocular symptoms,** consisting chiefly of papillary œdema, chemosis, and

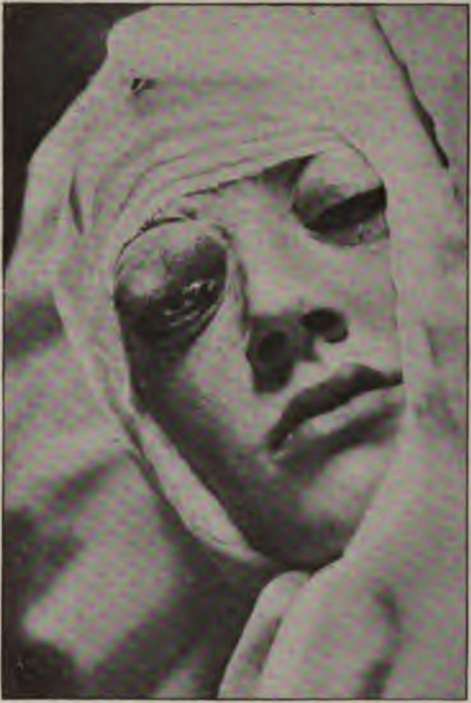

Fig. 100.—Œdema and exophthalmos in a case of thrombosis of the cavernous sinus. (*Sir StClair Thomson.*)

exophthalmos, to which may be added ulceration of the cornea and paralysis of ocular muscles from implication of the first division of the trigeminal nerve and of the motor nerves in their passage along the outer wall of the sinus. (Fig. 100.)

Swelling of the optic disc is also often found, but, if the case runs its course rapidly, many of the ocular symptoms do not develop.

The **prognosis** in a case of thrombosis of the cavernous sinus is very grave, and still more so if the condition of the eyes indicates bilateral disease. Suggestions and attempts have been made to reach these sinuses, but they have hitherto met with little success.

Thrombosis of the **longitudinal sinus** generally complicates some general debilitating disease and seldom arises from direct infection. It may be met with in weakened and wasted children, after typhoid fever, or it may complicate severe forms of anæmia.

The **symptoms** mostly depend upon interference with the cerebral cortex, and among them are mental dullness, convulsions, and possibly paralysis if the vascularity of the motor areas is modified. In many cases the symptoms are obscured by those of the previously existing general condition.

CHAPTER XLII

APHASIA

Etiology.—Aphasia is the term applied to speech defects of cortical origin, and it most commonly occurs as the result of a vascular disturbance, e.g. thrombosis, embolism, or—less frequently—hæmorrhage. Aphasia may also exist in association with tumours, and its presence may then be of assistance in localizing the seat of the growth.

Temporary attacks in which a patient has difficulty in speaking for a short time are not infrequently met with. They are probably caused by some passing vascular disturbance of the speech area of the cortex, or they may be caused by "diaschisis" (Monakow), i.e. by a temporary loss of function produced by the shock of a lesion some distance away.

When a lesion occurs in the course of the motor fibres below the level of the cortex, there may be difficulty in speech due to the inability of the impulses to pass down from the cortex, and in some instances this may amount to a complete motor aphasia, the power of understanding remaining unimpaired.

The views concerning the mechanism connected with the functions of speech are still in the stage of controversy. The older and, until recently, generally accepted theories which assume the presence of "centres" can most easily be explained by considering the education of a child.

The first step is the recognition of certain sounds which, by repetition, come to be associated with certain objects. Thus the memory for words is first developed in an area of brain cortex situated in the 1st temporal convolution of the left side.

After a time the child ceases to be content to hear words but wishes to produce them also, and, after repeated trials, succeeds in doing so by developing an existing (inherited) track to a portion of the cortex connected with movements of the tongue and lips. This part is situated in the posterior part of the 3rd frontal convolution of the left side, and is known as Broca's convolution. (Fig. 101.)

The child is now able to understand what is said and to make himself understood by others.

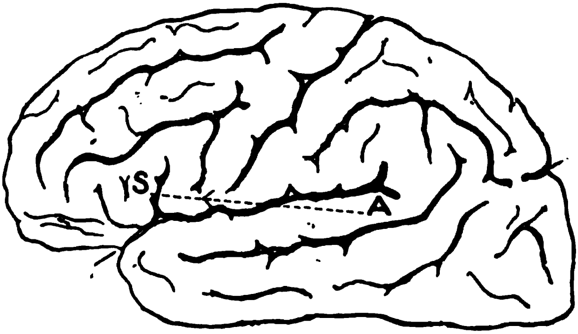

Fig. 101.—Motor-speech and auditory centres of the brain.

Further development takes place by teaching the child to read and write.

First the letters have to be recognized by educating the visual centre in the angular gyrus, and afterwards they have to be reproduced by the education of the writing centre, which is situated (probably) in the ascending frontal convolution close to the centre for other movements of the hand (Fig. 102).

We have now the two pairs of centres thus developed, and, to make the powers of communication still wider, further education makes it possible for impulses to pass from the auditory to the visual centres, and vice versa, so that the child is able to read aloud (in which the impulses pass from the visual to auditory, to Broca's convolution) and to write from dictation (in which impulses pass from auditory to visual and to writing centres) (Fig. 103).

In right-handed people those centres are, as a rule, all situated in the cortex of the left hemisphere, but in those who are left-handed the position is generally reversed.

Examination.—In examining a case of aphasia, the function of these four centres and the integrity of their commissural fibres should be tested in turn, and the following method is based on the plan adopted by C. E. Beevor:—

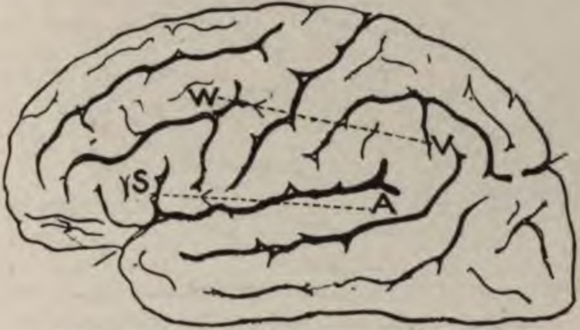

Fig. 102.—Visual and writing centres of the brain.

1. Test the **first pair of centres,** viz. the motor and auditory centres, and their commissural fibres.

(*a*) *The motor centre* (S).—Can the patient speak intelligently? This tests the condition of the motor mechanism at Broca's convolution. If this centre is destroyed he loses the power of coördinate speech, and is unable to repeat the names of words or objects though he may still be able to say a few words, usually the same ones over and over again. This condition is known as **motor aphasia.**

(*b*) *The auditory receptive centre* (**A**).—Can the patient understand what he hears? Speak some simple command, such as "Stand up," or "Hold up your hand." This tests the condition of the auditory receptive centre. If this centre is destroyed, sounds lose their meaning and convey no understanding—a condition spoken of as **word-deafness.**

(*c*) *Communication between auditory and motor centres* (**A–S**).—Can the patient repeat names of things or letters that he hears? This tests the condition of the fibres between the auditory and the motor centres.

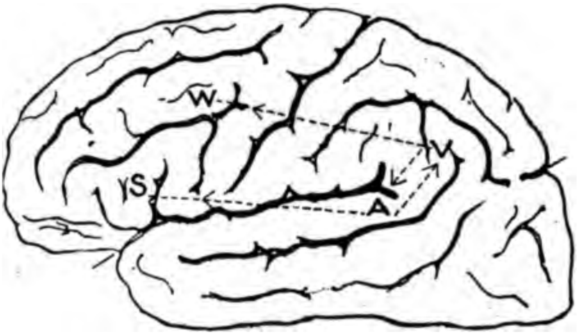

Fig. 103.—Auditory and visual centres of the brain.

2. Next test the **second pair,** viz. the visual and writing centres, and their commissural fibres.

(*a*) *The visual centre* (**V**).—Can the patient understand written language? Place before him some written request, such as "Stand up," or "Hold up your hand." This tests the visual centre in the angular gyrus, and, if its function is impaired, the letters, though seen, convey no intelligible meaning, and the condition is then spoken of as **word-blindness.**

(*b*) *The writing centre* (**W**).—Can the patient write spontaneously? This tests the centre for writing, which is probably situated near or in the area for that of movement of the hand. Failure is known as **agraphia.**

This test is often difficult to apply—first, because the auditory or visual centre has to be intact in order to convey the command; and secondly, because paralysis of the right hand, which is so frequently associated with aphasia, makes writing impossible. It is only very rarely that agraphia exists alone.

(c) *Communication between the visual and the writing centre* (**V–W**). Test the commissural fibres from the visual to the writing centre by asking the patient to copy from printed to written characters, which entails a visual impression that has been understood passing to the centre for hand movements.

3. Test the communication between the **auditory and visual centres**—from the auditory to visual, to writing centres; from the visual to auditory, to motor speech centre.

(a) *Communication between auditory and visual centres.*—Test the fibres which connect the auditory and the visual centre (**A–V**); for this purpose ask the patient to pick out letters or objects the names of which he hears. This entails the impulses passing from the auditory to the visual centres and being comprehended at both.

(b) *Communication from auditory to visual and to writing centres* (**A–V–W**). Test this by asking the patient to write from dictation.

(c) *Communication from visual to auditory and to motor-speech centre* (**V–A–S**).—Test this by asking the patient to read aloud and understand what he is reading.

If these tests for the different centres are applied methodically, noting after each where the patient succeeds and where he fails, some idea of the extent of the lesion will generally be obtained, but in practice the investigation is often made difficult by the partial or complete destruction of more than one area.

Individuals also vary considerably as regards the effects of partial lesions. Failure to read (**alexia**) especially seems to vary in this respect, and is no doubt often influenced by the degree to which the patient has been educated. Where a patient knows several languages, a comparison sometimes shows that he has retained more command over some than over others.

Also, from subcortical lesions, the impulses may be prevented from reaching a centre though the centre itself remains intact. If this should occur in the case of the auditory centre, the patient may be "word-deaf" though he can still talk, since his memory for words exists. Similarly, in the case of the visual centre, the patient may be unable to recognize printed words and yet be able to express himself in writing.

It is, of course, important not to confuse a peripheral lesion which impairs the articulation of language with aphasia.

While the conception of aphasia as outlined above has had its uses as a working hypothesis, it gradually became apparent that the schematic representations were too artificial and that, in practice, the symptoms of any particular case can seldom be made to correspond satisfactorily with the diagrammatic representations. Too much stress had been laid on the anatomical definition of "centres" and, in order to explain away the discrepancies, unsatisfactory attempts often had to be made to bring the symptoms into correspondence with the theories. In 1905 Pierre Marie expressed his dissatisfaction with these stereotyped views, and maintained that the idea of the existence of separate "centres" for the functions of speech could no longer be upheld, and that it was erroneous to believe that impulses conveying the elements of speech are fixed in special localities. To Marie, speech is an intellectual function for the adequate performance of which a particular portion of the brain is set aside, and consequently damage to this particular portion will produce more or less failure of speech as a whole rather than any particular elements of it—the degree, of course, varying according to the intensity of the lesion and the education of the individual. This idea is supported by the fact that in cases of partial aphasia it is the latest and highest acquirements that suffer first, just as it is in other branches of intellectual disorder. Thus, a patient who knows several languages may lose the power of expressing himself in those with which he is least familiar; and likewise, a man who knows only one language will find the greatest difficulty of expression with the more complicated parts of it.

Marie maintains that in every case of aphasia due to a cortical lesion there is some degree of intellectual impairment, as shown by the failure to comprehend spoken language, and that the omission to observe this fact hitherto has been due to the too simple character of the tests that have been applied.

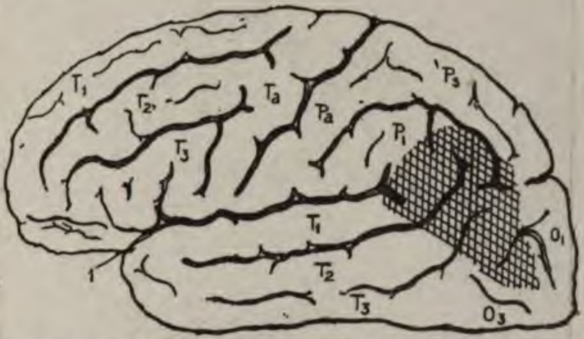

Fig. 104.—Lesion of angular gyrus and bases of two upper temporal lobes accompanied by aphasia of Wernicke. (*Pierre Marie.*)

The area which Marie considers to be the seat of this intellectual process is known as the zone of Wernicke, and comprises the supramarginal convolution, the angular gyrus, and the base of the 1st and 2nd temporal convolutions (Fig. 104).

The intellectual impairment which, according to Marie, is the essential factor of cortical aphasia may be accompanied by loss of power to speak if the lesion extends into and affects the conduction of the tracts of the subcortical region, a loss of power which he terms anarthria. Most striking of all, however, is his view that Broca's convolution plays no part in the production of speech, which view he supports by the citation of cases in which tumours and other lesions of this area have been unaccompanied by any signs of motor aphasia. (Fig. 105.)

It is not, of course, denied that aphasia is often associated with lesions of Broca's convolution, but it is maintained that a close examination of such instances shows that the destruction of tissue is not strictly limited to this area.

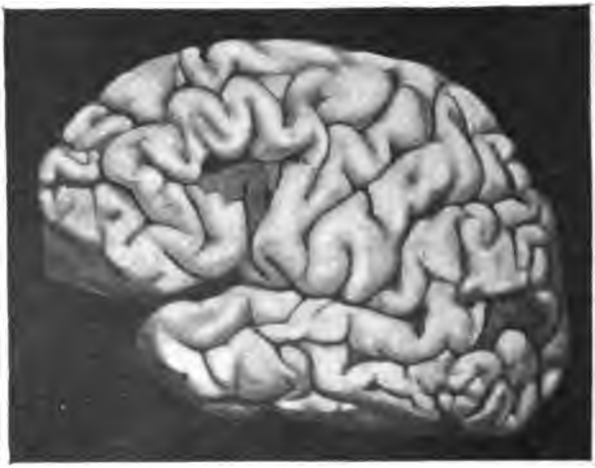

Fig. 105.—Cortical lesion at the level of the 3rd left frontal convolution in a right-handed man, without any disturbance of speech. (*Pierre Marie.*)

The three terms, Wernicke's aphasia, Broca's aphasia, and anarthria, are described by Marie as follows:

(1) In the **aphasia of Wernicke** the patients *can speak*, but their intellect is always impaired to a variable extent, so that they may talk nonsense; there is also loss of the power of reading and writing, and an incapability of comprehending spoken language.

(2) In the **aphasia of Broca** the patients can neither read nor write, neither can they comprehend fully all that is said to them. So far, they resemble the cases of aphasia of Wernicke, but they have the additional symptoms of being *unable to speak*.

(3) In **anarthria** there is simply an inability to speak from interruption of the motor fibres below the cortex. This disability is met with in association with lesions of the lenticular zone; and Marie maintains that the aphasia of Broca consists of the aphasia of Wernicke together with anarthria, and that it is not in any way connected with lesions of the third left frontal convolution.

And thus it comes about, as stated above, that Marie recognizes only one form of aphasia, viz. the aphasia of Wernicke, which he localizes in the supramarginal convolution, the angular gyrus. While Marie's views met with considerable opposition, more especially with regard to the exclusion of Broca's convolution, it was generally felt that an important advance had been made by raising the question and by suggesting a possible outlet from a position which was frequently difficult to reconcile with clinical facts.

Still more recently the subject has been entirely reopened by Head,* who has followed up suggestions originally made by Hughlings Jackson. As the result of elaborate investigations Head concludes that disorders in the use of language due to a unilateral cortical lesion cannot be ascribed to destruction of isolated "centres." He insists that in such disorders there is interference with the use of language as a whole, and though certain aspects of it may be disturbed more than others, according to the situation of the lesion, the defects of speech so produced cannot be expressed in simple terms of audition, articulation, vision, and writing. Head's view is that a cortical lesion causes dissociation of the highest forms of expression of thought (i.e. of symbolic thinking and expression), and according to the principal dissociations that have been observed to occur he has suggested the following provisional classification :

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* *Brain*, vol. xliii., pt. ii., 1920.

(*a*) *Verbal aphasia*, which is essentially a defect of word formation.

(*b*) *Nominal aphasia*, characterized by defective use of names and want of comprehension of the nominal value of words or other symbols.

(*c*) *Syntactical aphasia*, in which the patient tends to talk jargon.

(*d*) *Semantic aphasia* (σημαίνειν, to signify), in which there is a want of recognition of the full significance of words and phrases. The patient may understand each word or short phrase exactly as he can comprehend the details of a picture, but the ultimate meaning escapes him.

In most cases of aphasia two or more of these aspects of symbolic formation are affected, and in order to understand the nature of the disordered functions Head advises that examination should first be made of patients in whom the disturbance is as nearly as possible confined to one of these forms, after which the more complicated cases will be the better understood.

Treatment.—When there is reason to think that the aphasia is due to pressure on the cortical area for speech by tumours, extradural hæmorrhages, or other lesions of a removable character, it may be possible to obtain relief by a surgical operation.

When the functions of the centres have been permanently impaired, treatment should be directed towards re-educating the patient, by which means improvement, at any rate to some degree, may frequently be obtained. The chances of improvement are greatest in children, provided always that sufficient intelligence exists to make them capable of learning, and it is thought that possibly in some such cases the right hemisphere of the brain takes up the function after that for the left has been destroyed.

CHAPTER XLIII

MYASTHENIA GRAVIS (ASTHENIC BULBAR PARALYSIS)

The characteristic symptom of myasthenia gravis is the ease with which muscles become exhausted. Partial recovery of power often occurs after a period of rest, but the affected muscles are, as a rule, incapable of carrying out a sustained movement for any length of time.

Etiology and pathology.—The sexes suffer in about equal proportions, and in most cases the symptoms occur during the first half of life. There is still a considerable degree of doubt concerning the morbid conditions which underlie the symptoms.

So far as the central nervous system is concerned, beyond the occasional observance of some indefinite and inconstant changes in the nuclei of the cranial nerves, nothing notable has been recorded.

General opinion at the present time favours the view that the essential changes occur in the muscles themselves, and in support of this hypothesis is the finding by Weigert of an exudation of lymphoid cells in the muscle tissue, an observation which has been confirmed by Hun, Goldflam, Farquhar Buzzard, and others. It was present also in one of the cases which the author had an opportunity of examining.

Another abnormality often found is an enlarged thymus gland (Fig. 106) which is sometimes of a lymphosarcomatous character, but the connection, if any, existing between this and the exudation of lymphoid cells into the muscles is uncertain. Changes in the suprarenal bodies have also been described. At present one must be content to note the lesions and to assume that the disease depends either upon some modification of the internal secretions or upon some toxin of which the origin and composition are as yet unknown.

Symptoms.—The symptoms are brought about by failure of muscular power, but, as this is unequally distributed, they vary in different cases according to the parts affected.

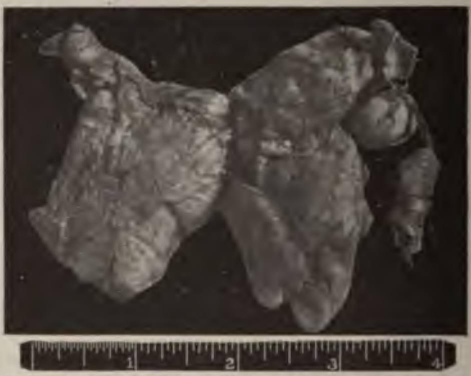

Fig. 106.—Enlarged thymus gland in a case of myasthenia gravis.

There are also great fluctuations in the intensity of the symptoms, alternate exacerbations and remissions being very characteristic of the disease. The "ocular" and "bulbar" distributions are common.

In the case of the **ocular symptoms,** ptosis is generally a prominent feature (Fig. 107), and the patients complain that the eyes cannot be kept open for long at a time. The lids persistently droop, and attempts are frequently made to correct the inconvenience by throwing back the head or even holding up the eyelids with the fingers. A night's rest often partially restores the power, which again wanes as the day wears on, and any sustained movement, such as looking upwards for any length of time, rapidly exhausts the muscles and brings on the drooping.

The compensatory action of the occipito-frontalis, seen in the wrinkling of the brow, which is a usual feature of ptosis from other causes, is generally absent, since the muscle is quickly exhausted by any attempts at continuous contraction. (Fig. 108.)

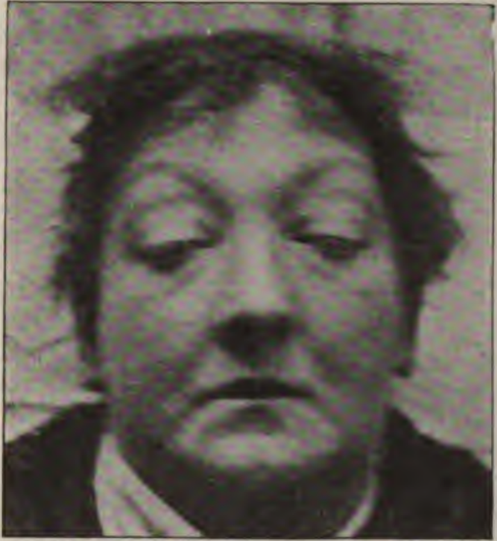

Fig. 107.—Bilateral ptosis in myasthenia gravis.

Transient ocular palsies of varying nature are common, and occasionally nystagmus is present.

The **bulbar symptoms** appear chiefly in connection with the orbicularis oris and the muscles of the tongue and palate.

Weakness of the orbicularis oris makes it difficult to blow or whistle, and also affects the speech.

The tongue when exhausted cannot be properly thrust out or forcibly protruded into the cheek. The paralysed soft palate ceases to shut off the nasal from the oral cavity, and so the nasal voice is produced, and at the same time there may be difficulty in swallowing, with regurgitation of fluids through the nose. All these muscles become weaker after use, and exhaustion of the masseters, which often takes place during mastication, may make it impossible to finish a meal. The facial muscles may also suffer, and the weakness of the muscles round the mouth often causes an alteration in the expression which closely simulates that associated with the myopathies.

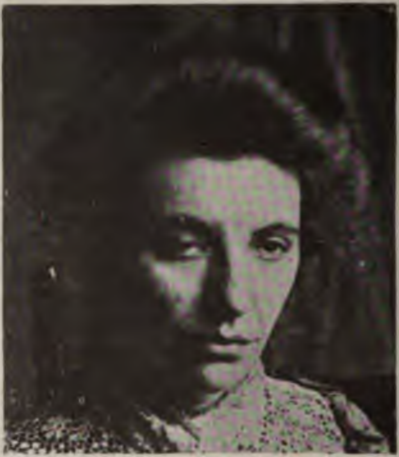

Fig. 108.—Ptosis and general facial expression in myasthenia gravis. Note the absence of any compensatory action of the occipito-frontalis. *(By courtesy of Dr. George Johnston.)*

Another characteristic is the "nasal smile," in which the movement at the corners of the mouth is deficient, the furrow of the smile being sometimes entirely above the upper lip (Gowers).

When the limbs are at fault, walking soon causes fatigue, and no occupation involving the use of the arms can be engaged in for any length of time. In the trunk, the muscles of the neck often suffer and the head falls forward on the chest. Weakness of the muscles of respiration causes dangerous attacks of dyspnœa.

Electrical reactions.—The rapidity with which the power of the muscles is exhausted by repeated contractions can be demonstrated by stimulating them with the faradic current. After a variable number of contractions have been induced, the response becomes gradually weaker and weaker, and finally ceases altogether, but may again be renewed after a period of rest. After the muscle has ceased to respond to the electrical stimulus it may still be capable of voluntary contractions. This condition is known as the *myasthenic reaction*, and when present is very characteristic of the disease; but, like the reaction of the muscles from voluntary movements, it varies from time to time, nor is it necessarily equally obvious in all the affected muscles at one time.

Reflexes.—The reflexes differ in their reactions according to the condition of the muscles which take part in their production. The knee-jerks are sometimes absent, or they can be exhausted by repeatedly eliciting them. Wasting of muscles is uncommon, but has occasionally been observed.

Sensory changes.—While the main symptoms are motor, there are occasionally some sensory changes to be noted. Aches and pains are liable to occur, and Farquhar Buzzard* has recorded cases in which the pains closely resembled in their intensity those of locomotor ataxy. The same observer has also shown that analgesia and anæsthesia may be found, and that the presence of mental symptoms is not unknown.

Diagnosis.—For purposes of diagnosis the cases of myasthenia gravis may be divided into three groups, viz. *ocular*, *bulbar*, and *spinal*, according to the parts in which the early symptoms predominate.

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* *Brain*, Part cxi.

1. **Ocular types.**—When ocular symptoms are the prominent feature, other conditions, in which ptosis and partial ophthalmoplegia externa are likely to occur, must be considered. In *tabes* when these symptoms are present there are almost certainly other distinctive signs, such as an Argyll-Robertson pupil, in association with lightning pains, loss of knee-jerks, and incoördination.

Transient ocular symptoms are sometimes associated with *migraine*, but here they generally bear some definite relation to the headache.

Cerebral tumours, *syphilitic disease*, and *hysteria* must also be considered as causes of ptosis. In hysteria the ptosis is usually spasmodic rather than paralytic, but outwardly it may show some similarity to that of myasthenia by the absence of the compensatory furrows of the brow.

2. **Bulbar type.**—When signs of bulbar trouble are prominent, *progressive bulbar paralysis* and *post-diphtheritic neuritis* will suggest themselves; in both these conditions the distribution of symptoms is apt to be similar. In progressive bulbar paralysis the early and pronounced wasting, with no remissions and with absence of the myasthenic reaction, will serve to enable the distinction to be made.

In postdiphtheritic neuritis the history and course are usually distinctive, and here again the electrical reactions will assist in the diagnosis. In very rare instances the diphtheria may cause permanent palsy of a bulbar type, cases of which have been recorded by Wilfred Harris.* The distribution of the paralysis in these cases very closely resembles that met with in myasthenia gravis, but, as Harris points out, the affection is usually limited to one or more of the bulbar nuclei, so that ptosis, and weakness of muscles of the jaw and of the neck and limbs, are not present, as they so often are in myasthenia. Moreover, there will be no variation of symptoms and no myasthenic reaction, while, on the other hand, there will be wasting and reaction of degeneration.

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* *Brain*, Part civ.

3. **Spinal type.**—Where vague symptoms of spinal distribution are complained of, such as fatigue after slight exertion, an erroneous diagnosis of *neurasthenia* may easily be made.

Prognosis.—The outlook in the majority of cases is, in the long run, bad, though remissions and intermissions of long duration may occur, and in a small percentage of cases the symptoms seem to have permanently disappeared. The tendency is for the patient to become more and more easily exhausted, and the danger to life lies chiefly in the occurrence of attacks of cardiac failure and respiratory dyspnœa.

Treatment.—In the absence of a definite knowledge of the cause of the trouble, efforts have to be directed towards preserving the contractility of the muscles. Rest, with the administration of strychnine and other tonics, is at present the best that can be done. The frequency with which an enlarged thymus gland has been found suggests the possibility of some modification or absence of an internal secretion as the cause of the disease, but hitherto the administration of glandular preparations has not been followed by any definite results

CHAPTER XLIV

CONVULSIONS

It frequently happens that a patient is seen for the first time on account of "fits." It is therefore important to review the general subject of convulsions, which outwardly may give no clue to their different causes.

It is convenient to divide the subject into two parts, viz. convulsions as they occur (1) in *childhood* and (2) in *adults*.

1. **Convulsions in childhood.**—Some of the principal conditions which are associated with convulsions during childhood are: rickets, idiopathic epilepsy, infantile hemiplegia and diplegia from various causes, meningitis, and cerebral tumours.

The commonest condition which underlies the convulsions of children is *rickets*.

The well-known tendency to convulsions in children is considered to be primarily due to an incomplete development of the central nervous system, so that the different parts are not under adequate control. Thus a healthy infant is always more liable to convulsions than a healthy adult, but should, nevertheless, if properly cared for, possess a nervous system sufficiently stable to enable him to pass safely through this period of life, unless exposed to some unusually severe strain, as, for example, the incidence of an acute specific fever. If, however, the naturally incomplete development is further retarded by neglect, rickets, or other circumstances, the stability will be still further lowered in varying degrees, and in that case comparatively slight variations in general health—intestinal disturbances, or other forms of irritation—suffice to bring on a fit.

Three types of children can be recognized in this respect :

(1) The healthy infant, in whom the relationship between different parts of the nervous system has not yet fully developed, but who still possesses a nervous system sufficiently stable under all ordinary circumstances.

(2) The child whose development has been abnormally retarded. Here, with care, convulsions may just be avoided, but the nervous system in this case is very lightly balanced, and a fit will readily occur from local or general disturbances.

(3) The child whose nervous system is so unstable that convulsions occur in spite of all care to avoid undue stimulation.

As the stability of the nervous system increases with the growth of the child the convulsions often cease, but in a certain number of cases this desired stability is never attained, and the child continues to be "subject to fits." It is this class which forms an important link between the convulsions of childhood and the epilepsy of adults, and between the two every gradation may be traced.

A recognition of these groups makes the part played by teething, constipation, flatulence, and other kinds of irritation in the causation of the fits easily understood. A healthy child may pass successfully through them all, but they will prove too strong for the possessor of an abnormally unstable nervous system and will start the impulses which give rise to the fit.

The convulsions connected with the other cerebral conditions mentioned above are incidents in the courses of the diseases and do not require separate description.

2. **Convulsions of adults** are chiefly associated with epilepsy, toxæmias (e.g. uræmia, chronic alcoholism, and poisons such as strychnine), degenerations (e.g. in

general paralysis of the insane), meningeal inflammations, cerebral tumours, hysteria, and onset of vascular lesions (hæmorrhages, thrombosis, embolism).

Reference to the chapter on Epilepsy (Chapter XLV.) shows that first attacks of this disorder become rarer as age advances, and therefore, when convulsions occur for the first time after middle life, suspicions should be entertained of the possibility of cerebral degeneration (especially general paralysis of the insane), cerebral tumours, and uræmia. It must, however, be remembered that patients who have taken a great deal of alcohol sometimes about this age have epileptiform fits which do not necessarily seem to signify the onset of any immediate cerebral degeneration.

In tumours of the brain a general convulsion, quite apart from the localized Jacksonian fit, is sometimes an early symptom, and may for a time be regarded as idiopathic epilepsy. It is therefore important to examine the discs of all patients who complain of fits of recent origin, even though it appears from absence of other symptoms that the fits are due to epilepsy.

The fits of the general paralytic may likewise be an early symptom of that disease, although they more often occur when the nature of the disease can be recognized from other signs. As far as the actual attack is concerned, it resembles that of an epileptic fit, either generalized or of a localized "Jacksonian" character; but whereas the ordinary epileptic generally shows no particular after-effects from the attacks, in the general paralytic every fit tends to leave the patient worse, both mentally and physically, than he was before. Sometimes apoplectiform fits, closely resembling those of cerebral hæmorrhage, alternate with the epileptiform seizures, which does not happen in ordinary epilepsy. It is thought that the fits in these cases are induced by the irritative properties of the products of the degeneration of nervous tissue taking place in the cerebral cortex. It is also probable that fits may sometimes be produced by a localized parenchymatous syphilitic degeneration which does not necessarily extend or lead on to general paralysis.

Uræmic convulsions occur occasionally as isolated symptoms, but are generally accompanied by definite signs of advanced renal disease. Sometimes the symptoms resemble those of cerebral hæmorrhage more than those of epilepsy.

Convulsions may occur in the initial stages of vascular lesions of the brain (hæmorrhage, thrombosis, and embolism), but the appearance of paralysis generally makes their origin clear.

Hysterical fits are described elsewhere (*see* p. 490).

CHAPTER XLV

EPILEPSY

An attack of epilepsy is characterized by a sudden and uncontrolled discharge of nerve energy which may give rise to all degrees of disturbance, from a momentary loss of senses or a transitory attack of giddiness to a severe convulsion. The negative results of an attack are seen in loss of consciousness (partial or complete), while the positive effects are shown in convulsive movements.

When all the cases of symptomatic epilepsy depending upon definite structural changes in the brain have as far as possible been excluded, there still remains a large group for which no adequate physical cause can be found and which are comprised under the heading of " idiopathic " or " essential " epilepsy.

Etiology.—Little is definitely known about the causation of " essential " epilepsy. In the majority of the cases the first attack occurs during youth, and where the fits seem to have begun in later life a careful inquiry will often reveal the previous occurrence of minor attacks which had passed almost unnoticed.

Convulsions during infancy, whatever be their cause, seem to dispose to epilepsy in later life, but there is often a considerable interval of freedom. Especial care, therefore, should be taken of children about the age of puberty, if they are known to have suffered from convulsions in infancy.

Epilepsy tends to run in neurotic families, in many of which there are also some members mentally afflicted.

In a person subject to fits any undue peripheral irritation may turn the scale, and in this way errors of refraction, nasal polypi, adenoids, ovarian and uterine disease, and other sources of irritation may, in certain circumstances, serve to start the explosion. In women there is occasionally some relation between the attacks and the menstrual periods.

Of recent years particular attention has been paid to psychological factors in the causation, and Pierce Clark attributes the underlying state to a congenital defect in the general make-up of the epileptic mind, with which are associated certain abnormal reactions, e.g. fits, disturbances of temper, lethargies and various psychic symptoms. Assuming the existence of such a primary defect, it can be understood how attacks might be precipitated by various stresses of life, more particularly at those periods when special demand is made upon the stability of the nervous system.

A consideration of *convulsions in general* seems to indicate that there are probably different groups of cases. In some it may be that poisons arising within the body may be powerful enough to cause convulsions (e.g. those associated with uræmia), and it is not uncommon to meet with convulsions indistinguishable from epilepsy as the result of chronic alcoholism. In such cases, however, the stability of the nerve cells has probably been previously lowered through structural changes, so that the fit is in reality symptomatic of nerve degeneration. In many cases it seems almost certain that the fault of the nervous system is chiefly local, as shown by the constancy and characters of the warning sensations at the beginning of the attacks, the spreading of which can be traced from one part of the cerebral cortex to another. Injuries to the brain, whether general or local, whether at birth or later in life, may be followed by fits which clinically are indistinguishable from those of idiopathic epilepsy. Abnormalities of internal secretions, e.g. of the thyroid and pituitary glands, are thought by some to be factors in causing epilepsy in certain cases, but the amount of influence, if any, which they exert is at present quite uncertain.

The mechanism by which the fit is produced is also uncertain. The main factors appear to be loss of control of the highest functional levels, accompanied by a discharge of nervous energy giving rise to the convulsion. That the one part may occur without the other is seen in "minor" attacks, where the highest levels of consciousness are often lost without the occurrence of any accompanying convulsive movements. There seems to be no doubt that the cerebral cortex is largely concerned in the mechanism of the attacks, as shown by the nature of the aura (*see* p. 436), but to describe the fits as being due to a "discharge from the cerebral cortex" is a crude statement which obviously requires qualification.

At present our knowledge may be summed up in the statement that an epileptic fit depends upon some want of adjustment between the functions of the cerebral cortex and the rest of the nervous system. In some cases, probably in many, it is the nerve cells that are at fault, so that toxins and stimuli are capable of producing a discharge quite disproportionate to their intensity; while in others the nerve cells may be of average stability, but the toxins reaching them may be too powerful to be withstood, just as the most stable of them will succumb to an over-dose of strychnine or to the poison of tetanus.

Symptoms.—Whether the cause is of local or of general origin, the main characteristic of an epileptic attack may be considered as a discharge of nervous energy as the result of a disturbance of function of the cerebral cortex. Hence the symptoms vary according to (1) the part in which the disturbance originates, (2) the direction in which it extends, and (3) the rapidity with which it spreads.

If a general view of the cerebral cortex is taken, it will be understood why warnings occur in some cases and not in others: how in some the disturbance of function is limited to a restricted area, and finally how in others a generalized convulsion results.

When the disturbance starts in any of the areas the functions of which are known, its first effects are those of stimulation of the given area. Thus a discharge beginning in the *occipital lobe* will give rise to visual symptoms, and in the *temporo-sphenoidal lobe* to auditory symptoms, and so on. If the fit begins in the *motor area*, twitching of a limb is often the first sign, just as it is when a gross lesion causes the irritation.

Thus the nature and position of the aura, which may be referred to almost any part of the body, indicate that particular area of the brain at which the disturbance of function has originated.

The different stages of a fit may be tabulated thus:

1. *Aura.*
2. *Convulsions:*
    (*a*) Tonic. (*b*) Clonic.
3. *After-effects:*
    Sleep, headache, and aching of limbs.
    Coma.
    Automatism.
    Temporary mania.
    Temporary paralysis.

At the termination or in the absence of the aura, the patient suddenly loses consciousness, and, with a respiratory spasm which often produces a groan or cry (the *epileptic cry*), falls to the ground with the muscles in a state of *tonic contraction*. In about half a minute this stage is succeeded by *clonic contractions*, which last for two or three minutes or so and then gradually pass off, leaving the patient usually in a deep sleep, or leading to one or other of the postepileptic conditions mentioned above.

The commonest *after-effect* is sleep of varying length, from which the patient is apt to wake with tired and aching limbs, as though he had been bruised all over. Sometimes the sleep deepens into coma,

The auræ.—These may be classified as sensory and motor.

1. **Sensory auræ.**
    - (*a*) Special senses:
        - Visual.
        - Auditory.
        - Smell.
        - Taste.
    - (*b*) General:
        - Sensations of something passing up a limb.
        - Tingling.
        - Giddiness.
        - Palpitation.
        - Flatulence and sensation in the pit of the stomach (epigastric aura).
    - (*c*) Psychical, e.g. dreamy states, reminiscences.

Auræ of special senses.—Of these the visual and auditory are common, while warnings of taste and smell are rare. Visual auræ often take the form of flashes of light, partial or complete loss of vision, and images of various kinds, while loud noises and whistlings are most frequently met with in connection with the auditory warnings. Sometimes words are heard, or a part of a sentence which may always stop abruptly at the same place in successive attacks, when consciousness is lost. Warnings of taste and smell seem always to be unpleasant; an indescribable "sulphurous" smell or a taste of "rotten cabbage" are among those described.

Auræ of general sensations.—The best-known is the sensation of "something passing up the arm or leg," from which the term "aura" was originally derived. The feeling generally begins in the hand and passes deliberately up the arm until it reaches a certain point, when consciousness is lost. With this kind of aura there is a good chance of arresting the fit by tying a cord round the arm and pulling it tight the moment the sensation is felt—a practical device which has been handed down from ancient times.

Most of these general sensations are vague and call for no special description, and in many of them the patients find it difficult to express their feelings in words.

Auræ of psychical sensations.—These warnings are interesting, and include various dreamy conditions which are often not altogether unpleasant. There also occur the peculiar sensations known as "reminiscences," in which there is a feeling of familiarity with a scene, as though it had all been seen before, though it is often impossible that there can have been any previous knowledge of it. These sensations may be experienced by healthy people, and have been well described by Dickens, Scott, Tennyson, Oliver Wendell Holmes, and other writers; but when they occur very frequently and vividly they should be regarded with suspicion, and careful inquiry should be made for any other symptoms, such as attacks of momentary dizziness, or the signs of spasm in the jaws, face, or eye muscles, which, if present, will indicate the need for treatment.

As a rule an aura lasts from a few seconds up to a minute or so, but sometimes it is of considerably longer duration, and enables the patient to try to check the fit and to get into a place of safety.

In about half the cases there is no aura at all.

Indefinite feelings, headaches, depression, and irritability sometimes enable the patient to prophesy the onset of a fit some hours, or even a day or two, before its occurrence.

2. **Motor auræ.**—These consist of twitching of parts of limbs, and where the extension of the discharge proceeds slowly its progress can be traced by the movements of successive parts of the limbs, just as may be done when a tumour causes the fit (Jacksonian epilepsy). It is important to emphasize the fact that the characteristics of Jacksonian epilepsy, where the convulsion spreads slowly from part to part, and either remains local, or becomes general with loss of consciousness, are by no means confined to cases where there is a tumour or other gross lesion (in which connection the

term was first used), but are very often found in association with attacks of "idiopathic" epilepsy.

Postepileptic automatism.—Here the highest cerebral functions are in abeyance and the patient performs coördinated acts without any knowledge of what he is doing: thus, he may walk for hours, and on coming to his senses may find himself miles from where he intended to go; or he may drive through crowded cities without any remembrance; or—most important of all—he may commit any kind of crime while he is in this irresponsible condition.

The important point in this connection is that the automatism is just as likely, or perhaps even more likely, to follow a minor fit which may be so slight as to pass unrecognized by those around, who therefore only see in the illegal act the impulse of the criminal. Mental disturbances sometimes appear to take the place of a fit, and are then spoken of as "psychic equivalents."

Postepileptic mania is not very common. It is often of temporary duration, but while it is present the patient may be very violent.

Postepileptic paralysis.—After a fit, one or more limbs are sometimes paralysed. The cause is presumably nervous exhaustion, and the power generally quickly returns. It is not a common occurrence.

Details of a fit.—It will now be instructive to follow out a fit in detail.

If the patient be subject to a visual aura the discharge will begin in the occipital area, which is the seat of the function of vision, and he has visual sensations which may take the form of bright lights or dimness of vision. The face will probably become pale.

The discharge, having once broken down the surrounding resistance, spreads with lightning rapidity over the motor and frontal regions. The part of the motor area which generally appears to feel the effect first is that connected with lateral movements of the head and eyes, and hence the head and eyes turn to one or other side (conjugate deviation). Almost synchronously with this movement the discharge spreads over the rest of the fore part of the brain. Consciousness is lost, and the intense stimulation of the motor area throws the whole body into a sustained spasm. The patient becomes quite rigid and falls like a log, uttering at the same time a cry, groan, or gurgling noise. He is now in the stage of tonic spasm, in which the muscles of respiration take part, and consequently the face becomes turgid and the veins swell. Frothy fluid appears at the lips. After a few seconds a little twitching is seen, beginning first probably in the face and then gradually becoming general; the limbs are alternately violently flexed and extended; the champing jaw often catches the protruding tongue, and blood is added to the frothy mucus in the mouth. The sphincters are frequently relaxed.

Then the violence of the movements gradually diminishes, until in two or three minutes or so all is quiet again, and the patient either gets up and goes about his business in a more or less dazed condition, or, if the circumstances are favourable, sleeps for several hours.

The conjunctival reflex is lost throughout the fit, and the pupils are dilated and do not react to light.

Immediately after a severe fit the knee-jerks may be temporarily abolished owing to the exhaustion of the pyramidal fibres consequent on the convulsion, but more often the knee-jerks are increased, and ankle-clonus and temporary extensor response of the plantar reflex may be present.

Status epilepticus.—In the status epilepticus, which is a rare complication of epilepsy, the patient has a rapid succession of fits, and in the more severe forms there are no intervals of consciousness between the attacks. It is always a serious condition, and in many cases death occurs from exhaustion.

Minor epilepsy.—Minor attacks are variously designated, either as "faints," "turns," or as "dizzy" and "peculiar" feelings. Apparently they are due to localized discharges of momentary duration, and their signs therefore pass off rapidly and are very limited in their distribution. The most common variety is characterized by a momentary loss of consciousness, thus indicating a disturbance of the highest functional level of the brain. The patient " loses himself " for a few seconds, stops abruptly in his conversation or work, perhaps drops something, and then continues as before. Such attacks often pass unnoticed, but, if attention is drawn to them, a passing pallor of the face, with possibly a slight twitching of some muscles, may be observed.

The " reminiscences " to which allusion has already been made are also common in minor epilepsy.

Visceral, auditory, and visual sensations are all common, and indeed any of the sensations which have been described as the aura of the major fits may occur as manifestations of the minor variety.

In this way the minor attacks may be looked upon as abortive fits in which the nervous disturbance remains strictly circumscribed. Presumably these slight disturbances are particularly apt to arise in the frontal region, since momentary loss of consciousness is such a common event. A patient may suffer from minor attacks for years before a major fit occurs ; in other cases both frequently occur, but in many the major attacks alone occur.

Postepileptic automatism, as already remarked, is very liable to follow a minor fit which may have been so slight as to pass unnoticed.

Diagnosis.—Epilepsy has to be distinguished from convulsions which have a definite organic or toxæmic basis.

From convulsions of infancy.—Little children are, as we have already seen, very liable to convulsions, which in the majority of cases cease to recur as development takes place. The individual convulsions are indistinguishable from those of epilepsy, but, bearing in mind this special tendency of childhood, it is permissible to hope that in any given case the fits will pass off as the child grows older. Suspicions may be aroused by the history of attacks in other members of the family, and by the fits occurring in an apparently quite healthy child without any adequate associated condition such as rickets.

Lastly, it must be remembered that a succession of convulsions in childhood undoubtedly makes for the occurrence of fits in later life, and it is common to find a recurrence of the convulsions about the age of puberty, even though there have been several intervening years of freedom.

From convulsions due to organic disease in children.—If the organic disease is acute, e.g. meningitis, the convulsions are, of course, for the moment, a symptom of minor importance. If the fits are associated with any of the forms of infantile hemiplegia or diplegia, or follow as a late result of meningitis, or from hydrocephalus, then they are likely to assume the ordinary character of epilepsy.

From convulsions due to cerebral tumour.—There are two conditions in which epilepsy may be confused with cerebral tumour:

1. *When the convulsion is general.*—Occasionally it happens that a general convulsion, indistinguishable from an epileptic fit, is the first sign of a cerebral tumour. While, of course, bearing in mind the possibility of other symptoms, the most certain way to avoid mistaking the generalized convulsion of a tumour for epilepsy is by examining systematically the optic discs. It is very likely that the appearance of the fundus will be suspicious, even if there are no other symptoms to suggest the true nature of the disease.

2. *When the convulsion is local.*—Tumours situated in the motor area of the brain cortex cause fits (Jacksonian epilepsy) which begin by localized movements that deliberately progress to other parts of the limb, and may or may not terminate in a general convulsion with loss of consciousness.

It must be remembered that many cases of ordinary epilepsy also begin in this way, and that in the complete absence of all other symptoms of a tumour the case is likely to be one of idiopathic epilepsy.

From convulsions due to general paralysis.—General paralytics are always liable to convulsions, and the possibility of these must always be remembered when a middle-aged man or woman suddenly has a fit for the first time. In some instances fits appear to be due to localized parenchymatous syphilitic changes in the cerebral cortex and, as such, need not be associated with any clinical signs to indicate the more widely spread changes of general paralysis.

From uræmic convulsions.—Uræmia sometimes first shows itself by a convulsion, but an examination of the urine will usually make the nature of the case clear.

From convulsions due to alcoholism.—Chronic alcoholics sometimes have for the first time in middle life a fit which appears to be epilepsy, though in many it is probable that the effects of renal disease are complicating factors.

From convulsions due to hysteria.—The difficulty of deciding whether a fit is hysterical is chiefly due to the fact that in most cases the medical man has only the history to depend upon.

The chief points on which reliance is placed in making the differentiation are as follows :—

	Epilepsy	Hysteria
Cry	A single cry or groan...	Often several shrieks.
Fall	Sudden, without any attempt to save themselves	Slide down; seldom hurt themselves by falling.
Sphincters ..	Often relaxed	Not relaxed.
Tongue ...	Bitten	Not bitten, but lips may be.
Struggling ...	No purposive struggling	Often struggle and fight, requiring several people to hold them
Presence of others	Fits occur when patient is alone or in presence of others	Usually in presence of others.
Duration ...	A few minutes ...	Often a long time.

It is necessary to remember that an hysterical fit may directly succeed one of epilepsy, and that the latter may be of the "minor" kind, and be quite unnoticed by the ordinary bystander.

Prognosis.—It is difficult to estimate with any accuracy the number of cases in which fits have entirely ceased, for such patients naturally escape further observation. It is generally thought that the disease is arrested in from 5 to 10 per cent. of all cases. It is impossible to predict what course the disease is likely to take until the effects of steady treatment are known, for it is rare for the fits to disappear spontaneously.

Long intervals between fits, and absence of minor attacks, are both favourable points, and cases beginning in later life are generally more easily influenced than those arising about the period of puberty. Statistics show that the prognosis is no worse in cases where there is an hereditary tendency. The principal danger to life, except when the rare condition of status epilepticus occurs, arises from possible accidents, of which the chief are suffocation from turning on to the face in nocturnal epilepsy, injuries or death from a fall when consciousness is lost, and choking from the passage of vomited food into the larynx. In a considerable number of confirmed epileptics there is mental deterioration, which not infrequently becomes advanced enough to constitute insanity.

Treatment.—To treat epilepsy with any degree of success the utmost attention must be given to details.

The daily life should be arranged to avoid any conditions which are likely to cause sudden variations in the equilibrium of the nervous system.

Peripheral stimuli of undue strength from without, as the sudden appearance of bright lights, loud noises, and other things causing "starts," must be avoided just as much as those from within, and a quiet, even life, with perfectly regular hours, should be led. The occupation should be physical and mental, judiciously proportioned to each case. It is most important not to allow idling and day-dreaming. Recognition of the possibility of psychological factors in the causation of epilepsy has further emphasized the importance of broad educational treatment as a means of improving the general mentality of patients and increasing their powers of adaptation to the general stresses of life. All evidence shows that occupation reasonable in kind and degree is beneficial, and it is largely due to the opportunity of leading a mechanical life, with proper occupation, that epileptic colonies are so successful. It is not common for epileptics to have fits when they are engaged in some engrossing occupation; the dangerous moments seem rather to be those in which the mind is aimlessly wandering, and just between the sleeping and the waking states is a favourite time for a fit.

Arrest of fits at their onset.—Many patients find out for themselves that they can frequently ward off a threatening fit by "force of will." This power should be encouraged, and it may often be aided by muscular exertion. The moment a fit threatens, the patient should clench his teeth and grasp something tightly. The most convenient method is to clasp his own arm as tightly as possible, and in many instances the discharge can in this way be arrested.

The possibility of arresting fits by tightening a cord when the aura passes up a limb has already been mentioned (p. 436), and, when practicable, should always in such cases be employed. Smelling strong salts and inhalations of amyl nitrite sometimes meet with success. Every fit stopped is a distinct gain, and where the fits are few and far between it is most important to make the gaps as wide as possible.

Diet.—The food should be simple and quite plainly cooked, and spices of all kinds should be avoided. The question of allowing meat is one on which there is some difference of opinion. Certainly, the meat taken should be moderate in amount and of an easily digested nature, but there seems to be no great benefit in abolishing it altogether, at least in the majority of cases. More recently it has been thought that the harmful constituents of food are the "purin" bodies,* and diets have been constructed to eliminate these. Such a diet would include milk, eggs, butter, cheese, rice, macaroni, tapioca, white bread, cabbage, lettuce, sugar, and fruit, while in potatoes and onions the amount of purin bodies is very small. Tea, cocoa, coffee, meat, and fish should, on this hypothesis, be avoided. Among the meats which contain the least quantities of purin bodies are tripe, neck of pork, and cod-fish.

Aldren Turner† has found that diets constructed on this principle seem to be beneficial in some instances, but, like other forms of treatment, are not likely to produce an effect that is constant for all cases.

Elimination of salt.—The elimination of salt from the diet has been advised on the supposition that the bromide can take its place in the tissues of the brain. By this means it was hoped that the fits might be controlled by smaller doses of bromides, which at the same time would have a more powerful effect. No constant results have been obtained, but a certain proportion of cases seem to have benefited, so that it is often worth a trial.

Drink.—Alcohol in any form should be absolutely forbidden. Coffee is too stimulating; but weak fresh tea may generally be taken. Water and milk are the best drinks for general purposes.

Bowels.—The bowels should be most carefully regulated. Constipation very often appears to be the exciting cause of an attack.

Drugs.—The bromides of potassium, sodium, and ammonia, given judiciously, are the most useful drugs in epilepsy, and, in spite of its more depressing influence, potassium bromide is the one which appears on the whole to give most satisfaction. Many prefer to give the different bromides together. Bromide of strontium also is useful.

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* The purin bodies are substances of which the composition C_5N_4 forms the basis; they are found in many common foods, and also as the result of tissue metabolism of the body. See "Purin Bodies of Food-Stuffs," by Walker Hall.

† *Practitioner*, April, 1906.

Regulation of dose of the bromides.—Much of the success in treatment depends upon carefully regulating the doses. If too little is given, the fits are not checked; while if the amount is unnecessarily large, mental depression and dullness are produced. The ideal dose is that which is just sufficient to check the fits, but it is, of course, impossible to lay down any rules, as so much depends upon the effects—good or bad—of bromides in every individual case. Broadly speaking, the dose limits of bromides in epilepsy are between 10 and 80 grains a day. Larger doses are sometimes needed, but as a rule are not beneficial when the smaller amount has no good effect.

Time for giving bromides.—Much may be done by considering the times at which fits usually occur in relation to the taking of medicines. Where the attacks show no regularity the doses may be taken at convenient intervals twice or three times a day as required, but where it is possible to calculate to some extent on the onset of a fit, a judicious anticipation will often meet with success. Thus, if the fits occur only during sleep, or in early morning, a single dose of 20 or 30 grains of bromide may be taken the last thing at night.

Drawbacks of bromides.—The drawbacks to taking bromides continuously are the tendency to the production of (1) acne, (2) mental depression.

The tendency to acne can be counteracted by combining small doses of arsenic with the bromide. This in its turn has a drawback, for arsenic taken over a long period of time may give rise to pigmentation of the skin. When combined with bromide the arsenic in the doses usually given has not the same tendency to produce neuritis or other unpleasant effects which it is sometimes apt to do when taken in other ways.

An example of a prescription is as follows :—

℞ Potassii bromidi	Dg 12	gr. xx
Liquoris arsenicalis	Cl 12	♏ii
Spiritus chloroformi	Ml 1	♏xv
Aquam	ad Ml 30	℥i

Two tablespoonfuls to be taken twice or three times a day after food.

As already mentioned, the bromides of sodium or ammonia may be substituted for that of potassium, or the dose may be divided between the three.

Adjuvants to bromides.—While bromides form the basis of nearly every prescription for epilepsy, there are other drugs which, when combined with them, appear often to have some good influence. Of these the most important are belladonna and borax, either or both of which may be combined with the bromides

℞ Potassii bromidi	Dg 12	gr. xx
Boracis purificati	Dg 3	gr. v
Tincturæ belladonnæ	Dl 3	♏v
Liquoris arsenicalis	Cl 12	♏ii
Spiritus chloroformi	Ml 1	♏xv
Aquam	ad Ml 30	℥i

Two tablespoonfuls to be taken three times a day.

Occasionally the addition of small doses of digitalis and potassium iodide seems to have a good effect. Iron may be of assistance when there is anæmia, but in some cases it disagrees. Nitro-glycerin may, in the form of liquor trinitrini, be given with the bromides when the condition of the peripheral circulation seems to warrant the administration of vaso-dilators. Sodium luminal (a derivative of veronal) has recently been used with good results in some cases. A usual dose is 1 gr. for an adult and ½ gr. for a child, twice a day.

The minor fits are often more difficult to control than the major ones; and where bromides fail, pills of zinc oxide may be tried. An endless number of drugs has naturally been tried and commended at various times, but up to the present no mixture has met with such success as those of which bromides constitute the basis.

Surgical treatment, often useful where fits depend upon a gross lesion, is very seldom indeed of use in general epilepsy. Here and there a very local and deliberate onset may suggest the advisability of removing a piece of the cortex, but in the large majority of cases no operation is likely to produce a cure.

Summary of treatment.—The points to which attention should chiefly be given may be summed up as follows :—

1. General mode of living.
2. Diet.
3. Alimentary canal.
4. Removal of all sources of peripheral irritation.
5. Administration of drugs.
6. Education and, in suitable cases, psychotherapeutic treatment.

CHAPTER XLVI

TETANY

TETANY is characterized by spasmodic tonic contractions of the muscles, chiefly those of the extremities, and by an increase in the irritability of the muscles to electrical and mechanical stimulation.

Etiology.—Tetany is most commonly to be seen in children who are the subjects of rickets. More rarely it occurs in adults, and has been noted in association with chronic dilatation of the stomach, diarrhœa, debility after lactation, and removal of the thyroid gland, in connection with which the parathyroid is supposed to be the essential factor. Occasional epidemics have been recorded.

Pathology.—The contractions are probably set up by a toxæmia which arises at some time during the course of the diseases mentioned above. No definite lesions have been found after death, and the exact seat of the irritative process, whether in brain, cord, or nerves, is a matter which still remains undecided.

Symptoms.—The spasms, which are often preceded by sensations of tingling and numbness, are generally most prominent in the distal parts of the extremities. The hand usually assumes an attitude which is characteristic. The fingers are flexed at the metacarpo-phalangeal joints and extended beyond, a position which is brought about by a spasm of the interossei, while the thumb is brought into the position of opposition and pressed tightly into the palm. At other times the fingers assume the shape of the "claw-hand." The wrist- and elbow-joints are flexed, and the arm is adducted. Tonic spasms of a corresponding nature occur in the feet; the foot is inverted and the toes are flexed. Swelling of the hands and feet sometimes occurs, and the attacks may also be accompanied by a considerable amount of pain and tenderness in the limbs. In severe cases the spasms spread to the trunk and cause opisthotonos, and in some instances the tongue and muscles of the face are affected.

The attacks last for variable times. Sometimes the spasms pass off in a few minutes, and at others they are more or less continuous for some hours.

Trousseau observed that an attack may be brought on by pressure on the main nerves and vessels of the limb.

The muscles and their motor nerves are very irritable to mechanical stimulation (Chvostek's sign), a fact which can be well observed in connection with the muscles of the face. The sensory nerves also are unduly irritable.

The excitability of the nerves to electrical stimulation, especially to that of the galvanic current, is greater than normal, and the anodal opening contraction is greater than the kathodal closure contraction.

Diagnosis.—The carpo-pedal contractions of a typical case are not likely to be confused with anything else.

When the spasms extend to the trunk and face, the possibility of tetanus may present itself, but is negatived by the order in which the contractions have appeared, as well as by the absence of trismus, which is such a characteristic early sign of tetanus.

Prognosis.—The occurrence of tetany in children is not usually dangerous, but sudden death has occasionally taken place in association with the spasms. As a complication of chronic dilatation of the stomach, tetany may be very troublesome or even dangerous; and if organic disease of the stomach be present, the onset of the spasms may indicate the immediate necessity of obtaining some relief through a surgical operation.

Treatment.—In children the bowels should be relieved by castor oil, and enemata if necessary. Warm baths may be useful during an attack. Among the sedatives that are most likely to bring relief are bromides and chloral. Attention must also be paid to the general health, which in the case of children is usually impaired by rickets.

CHAPTER XLVII

PARALYSIS AGITANS (PARKINSON'S DISEASE)

Paralysis agitans is characterized by the gradual onset of weakness, tremors, and rigidity.

Some degree of weakness is probably present in all cases, but the amount of tremor and rigidity is very variable.

From the point of view of diagnosis, rigidity is quite as important a symptom as tremor, and the nature of the disease is often clear though there may be no tremor.

Etiology.—It is not possible to attribute the onset to any direct cause, though injury, shock, worry, or anything that causes general debility sometimes appears to be the starting-point. Between 45 and 60 is the age at which the disease usually begins, and men are rather more commonly affected than women. Symptoms indistinguishable from those of paralysis agitans have recently been observed as occasional results of encephalitis lethargica (*see* p. 344).

Pathology.—Nothing definite is known concerning the underlying changes in the nervous system. Alterations in the structure of the cortical cell bodies and their dendrons have been found by some, while others have described arterial and perivascular changes; but none of these seems to be constantly present. It has been suggested that the modification or want of some internal secretion, more especially of that connected with the parathyroids, may be the cause. Others consider that the essential changes take place in the muscles. Wilson has remarked upon the resemblance that exists between the symptoms of paralysis agitans and those of progressive lenticular degeneration (*see* p. 386). Tremors, rigidity, and weakness are common to both conditions, and in these circumstances Wilson considers it might be expected that the localization of the lesions should be more or less identical, i.e. in the corpus striatum. Jelgersma is quoted as having demonstrated degeneration of strio-thalamic fibres and of the lenticular bundle of Forel in paralysis agitans.

In any case, the frequency with which the onset of symptoms assumes a hemiplegic distribution is strongly suggestive of the cerebral origin of the disease.

Symptoms.—The onset is usually insidious. Occasionally the tremor seems to date definitely from the time of an injury or shock, but more often it begins so gradually as to be scarcely noticeable.

In some a slow, progressive weakness, with increasing stiffness, marks the onset; while in others the weakness, tremors, and rigidity all appear at about the same time.

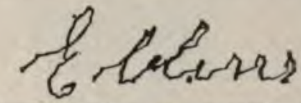

Fig. 109.—Writing in a case of paralysis agitans.

The *tremors* are generally first noticed in one hand; the thumb and forefinger move rhythmically in a way which has been aptly described as "pill-rolling" in character. At first the trembling is scarcely noticeable but gradually obtrudes itself until it begins to interfere with ordinary actions such as writing. (Fig. 109.)

From the forefinger and thumb the tremor spreads to the other fingers, and at last the whole hand takes part in the movement, which later may extend to the elbow and to some extent to the shoulder.

By an effort of will the movements can be controlled, but only for a few seconds, and in severe cases an attempt to keep one limb still generally results in increased movements of another.

The tremors are most obvious when the limbs are at rest, and, indeed, during brief voluntary movements, e.g. holding out the hand, they become distinctly less, though they do not altogether cease, as is sometimes stated.

This remission during voluntary movement can be easily demonstrated by asking the patient to write or draw a line, an action which, from the tremor present, would often appear almost impossible, but which on trial often proves to be done comparatively well (Fig. 110).

The tremors cease during sleep.

From one hand the tremor generally spreads to the leg on the same side, and thence to the hand on the other side, but there are many exceptions to this rule. Some of these are more apparent than real, for the tremor in the legs is often less noticeable than that in the arms, probably because the muscles are more in action—movement, as already mentioned, becoming more obvious when the muscles are relaxed.

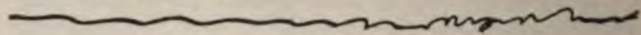

Fig. 110.—Line drawn by a patient with a severe tremor, showing at the beginning power temporarily to control the tremor.

As the disease progresses, the head, face, and jaws may all take part in the movements.

Rigidity is even more characteristic of paralysis agitans than tremor.

At first scarcely noticeable, it creeps on relentlessly until the figure becomes firmly set in the characteristic position (Fig. 111). Obeying the usual laws, the flexors overbalance the extensors, and the patient becomes bowed like an old man, with the head bent forwards and the body flexed The arms are adducted at the shoulder and flexed at the elbows, while the fingers and thumb are bent into a half-closed position. The thumb is often curved in a position of hyperextension (Fig. 112).

The *features* are *fixed* and expressionless (*Parkinson's mask*). The voice is dull and monotonous, owing chiefly to the rigidity of the muscles of the lips and face.

It can easily be imitated by trying to talk without moving the lips.

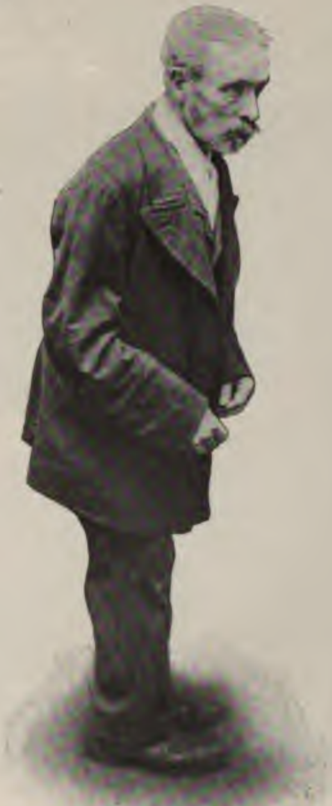

Fig. 111.—Case of paralysis agitans showing the characteristic attitude.

Voluntary movements are performed slowly and stiffly, and the reaction times are thereby lengthened.

The relaxation of muscles also takes place more tardily than usual.

The rigidity of the bowed body with the upper part bent over throws the centre of gravity forward and leads to the peculiar *gait*, in which the patient takes short, rapid steps that oftentimes amount to a run, and when once started he has considerable difficulty in stopping. From his appearance it has been well said that the patient looks as if he were trying to catch up his centre of gravity.

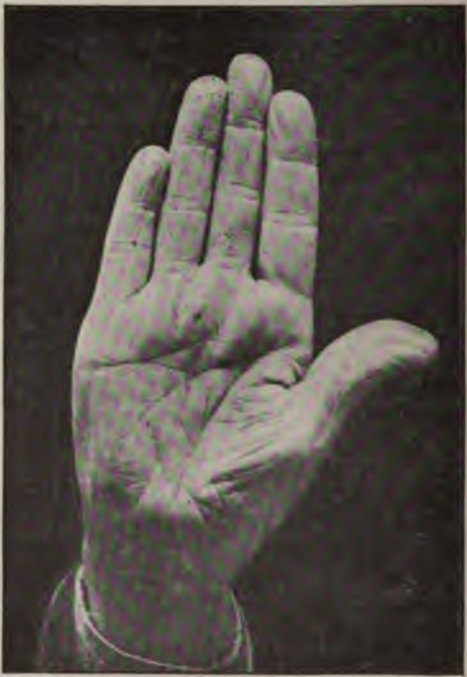

Fig. 112.—Hyperextension of the thumb in a case of paralysis agitans.

The mechanism of this peculiar gait can be best understood by imitating the conditions under which it occurs. If the body is bent well over and kept stiff, so that the balance is only just preserved, and a short step forwards is taken, there will be a tendency to fall, which is only counteracted at each step by rapidly shuffling the hind foot forwards. This movement is repeated indefinitely, and the patient being unable to bend the body back, and so to restore the balance, there is difficulty in stopping without running the risk of falling, unless he catches hold of something for support.

In the same way, if the patient is suddenly started backwards, the rigidity of his body makes it difficult to regain the balance, and the movement backwards is involuntarily continued for some steps. This is known as *retropulsion*. A similar effect can also be obtained by suddenly starting the patient on a side movement (*lateropulsion*).

The *deep reflexes* are generally said to be unaltered, but in many cases, especially before rigidity becomes marked, some exaggeration may be found. Likewise, with regard to the plantar reflexes there is usually no alteration, but systematic examination has shown that every now and then an instance occurs in which a typical extensor response is obtained. Taken in conjunction with the view already mentioned, that the seat of the disorder is in the corpus striatum, this behaviour of the reflexes is interesting, since it shows that the degeneration, although primarily outside the pyramidal motor tracts, may occasionally extend into them, and so cause exaggerated knee-jerks and extensor responses to be added to the usual symptoms of the disease.

Among other symptoms are *cramps*, which are often troublesome. Some patients complain of the toes drawing up under the feet and troubling them in walking.

Sensations of *heat* and *cold* are common, and are apparently of vaso-motor origin.

In the later stages, when it is almost impossible to move voluntarily, there is often very great restlessness, with a desire to have the position of the limbs constantly altered.

Prognosis.—The disease progresses slowly, and finally the patient may be quite bedridden. There is no special danger to life.

Treatment.—The only chance of doing anything for these cases is to begin treatment early. Even then it is only palliative, but undoubtedly much comfort and some retardation of symptoms can often be obtained by judicious treatment.

This has to be directed towards relief of the three main symptoms, tremors, rigidity, and weakness.

The cases in which the tremors are severe are the most painful, for the patient is never able to be at rest, and, unfortunately, no drugs can be depended upon to still them. All the usual sedatives may be tried, but only seldom is any lasting effect produced. Opium and its preparations are of no use.

When the tremors are very bad, hyoscine sometimes checks them to some extent. It may be given in doses beginning with $\frac{1}{200}$ gr. of the hydrobromide, but it must be used with caution on account of its toxic effects; and it cannot be said that any great relief is often obtained from this or any other drug.

In slight cases the patient should be encouraged to control the tremor, but in the more severe any sustained effort is very exhausting. Little devices of various kinds can sometimes be found to give comfort. In one patient, carrying a heavy stick gave some relief to the tremor.

It is scarcely necessary to state that as quiet a life as possible should be led. Excitement makes the tremor worse, and in some instances, so long as the patient remains quiet, it gives him very little trouble for quite a number of years.

The rigidity is best combated by carefully regulated active and passive movements, and, if these are practised early enough, there is no doubt that the tendency to permanent fixation may be postponed. Hot baths regularly given and accompanied by light massage also afford relief.

The weakness requires no special form of treatment. The main point is to keep up the general nutrition: the diet should be suitable in quantity and quality; and the patient should live as much as possible in the open air—a mode of life which seems to exercise a general influence in retarding the progress of the disease.

CHAPTER XLVIII

CHOREA (RHEUMATIC CHOREA, SYDENHAM'S CHOREA, ST. VITUS'S DANCE)

CHOREA is characterized mainly by the presence of involuntary movements, together with varying degrees of muscular weakness and incoördination. Frequently there are also some mental changes present.

Etiology.—Chorea is a disease chiefly of *children* between the ages of 5 and 15, and occurs more often in girls than in boys.

Rheumatism is undoubtedly the principal underlying cause, and statistics show that a definite history of rheumatic manifestations can be obtained in a very large number of cases. Less often an attack of rheumatism follows chorea, and only occasionally are the two conditions associated.

Acute specific fevers other than rheumatism are not directly concerned in the causation of chorea. The apparent relationship of the disease to scarlet fever may depend upon the close association which exists between the latter affection and rheumatism.

In the rheumatic child anything which strains the nervous system seems to dispose to an attack of chorea; hence inherited nervousness, too much schooling, frights, errors of refraction, and other forms of irritation often appear in the history of the case as the apparent causes. Fright is especially apt to be closely followed by chorea.

Chorea is sometimes associated with *pregnancy*, but in the majority of these cases also some history of previous rheumatism can be obtained.

Pathology.—That this form of chorea is a manifestation of rheumatism is now generally conceded. Poynton and Holmes* consider that the *Diplococcus rheumaticus*, the presence of which they have demonstrated in the pia mater of fatal cases, is probably the infective agent, and among the microscopic changes that have been observed are hyperæmia, thrombosis, swelling and chromatolysis of the cortical cells.

Clinical signs which indicate the presence of organic changes in the nervous system have also been frequently observed, so that the disease cannot any longer be classified among those of "functional" origin, but, as suggested by Grenet and Loubet,† must rather be regarded as a form of infective encephalitis.

Symptoms.—The onset is often insidious, and commences with "inattention," "dropping things," and "fidgeting." Sometimes, especially after a severe emotional disturbance, the onset is acute, and either weakness or irregular movements appear quite suddenly.

The symptoms may be divided into the following groups :—

1. Involuntary movements.
2. Muscular weakness.
3. Incoördination.
4. Mental changes.
5. Associated rheumatic manifestations.

1. **The movements.**—The movements are spasmodic, involuntary, and irregular. They may be distributed all over the body, or they may be confined to one limb or to one side (*hemichorea*). Their intensity varies in degree : in one case it may be quite impossible to keep the patient in an ordinary bed ; in another the movements can only be detected with difficulty. In slight cases the movements generally become more obvious if the child holds out both hands with the fingers widely outspread, and at the same time protrudes the tongue.

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* *Lancet*, Oct. 13, 1906.

† *Revue Névrologique*, Dec. 15, 1912.

Irregularity is a characteristic of the movements, and the same movement is never repeated continually, as it is in the tics. The hands are generally more severely affected than the upper arm, and when the fingers are spread out there is a tendency to hyperextension. Peculiar grimaces are made, which, together with spasms of the tongue and jaw, often make talking and eating difficult.

The diaphragm may contract spasmodically, and the heart may beat irregularly. Excitement of any kind makes the movements worse, and in severe cases the whole body is tossed about with great violence. During sleep the movements disappear.

2. **Loss of power.**—There is probably some weakness in almost every case of rheumatic chorea, but it is often difficult to differentiate it from the spasmodic movements and incoördination, both of which cause apparent loss of power in the holding of objects.

Loss of power in one or more limbs is, however, an important feature in a considerable percentage of cases, and occasionally it is the predominating one, in which case the term *paretic chorea* is sometimes used. At first sight such cases may simulate the paresis of acute anterior poliomyelitis.

3. **Incoördination.**—There is considerable difficulty in carrying out voluntary movements with precision, as is easily seen by asking the child to perform some delicate act, such as picking up a tiny object, when the fingers move irregularly round it before it is finally seized.

4. **Mental changes.**—Dullness, want of attention, and inability to do lessons are often noticed. In bad cases, especially when it is difficult to feed the child, sleeplessness may be a troublesome symptom. In the severe cases there may be acute delirium accompanied by a high temperature.

There are no definite sensory changes peculiar to chorea. Occasionally there is some degree of anæsthesia of varied distribution, and the disease may sometimes be further complicated by the presence of hysterical manifestations. In some cases, too, complaint is made of pains in the limbs.

5. **Associated rheumatic manifestations.**—Endocarditis is the most important rheumatic manifestation that frequently accompanies chorea; the mitral valve is the one that is usually affected, and a systolic murmur can generally be heard at the apex. In fatal cases, bead-like vegetations of recent origin may be found upon the cusps.

Erythema nodosum may be present in conjunction with the chorea.

Joint troubles are more likely to have occurred at some previous time, but occasionally they are met with during the attack.

Careful examination frequently reveals the presence of signs that are usually associated with organic changes in the central nervous system.* Some, e.g. an extensor response and hypotonus of the muscles, are indicative of lesions of the pyramidal tracts, while others, as the inability to perform successive movements such as pronation and supination of the forearm rapidly (*adiadochokinesis*), and failure to arrest the movement of the finger at a given spot (*asynergia*), suggest a deficiency of the functions of the cerebellum.

The irritative nature of the infection is further shown by lymphocytosis, which has sometimes been observed in the cerebro-spinal fluid after lumbar puncture.

The knee-jerks may be difficult to obtain, but when elicited they are often rather exaggerated. Sometimes, owing to the irregular contraction of the quadriceps extensor, the leg hangs in the air longer than usual, a condition which has been termed the "sustained knee-jerk."

The sphincters are usually unaffected.

Diagnosis.—The irregularity and persistence of the movements serve to distinguish chorea from the *tics.* In the tics, periods of complete repose alternate with exacerbations (p. 468).

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* Grenet and Loubet, *op. cit.*

The *athetosis* of hemiplegia or diplegia in children is a much slower movement, and, moreover, the persistence of the movements and the permanent paralysis, which is often accompanied by arrested mental development and epilepsy, all point to the nature of the case. The history is also frequently an aid in diagnosis, for in most cases it is found that the paralysis (if not the athetosis) and the mental changes date from birth, or have supervened shortly after at an age at which chorea never occurs.

Hysterical movements may closely resemble chorea, but, like those of the tics, they are usually more regular. In cases of hysteria other confirmatory manifestations will usually be found, but it must not be forgotten that it is possible for both conditions to be present at the same time.

In *myoclonus* the spasms are shock-like. The trunk is more liable to be affected, and the contractions tend to be symmetrical.

Prognosis.—Patients with rheumatic chorea generally tend to get well in the course of a few weeks, but the progress is often slow, the affection lasting, on an average, between four and twelve weeks. There is a strong tendency for the patient to relapse just when the symptoms seem to be abating, and there is also a distinct tendency to recurrence after varying intervals.

Exceptions to this benign course occur every now and then. Severe cases in which the body is incessantly thrown about may end fatally with symptoms of exhaustion, delirium, and hyperpyrexia, and to such the term *chorea gravis* is applied.

Endocarditis which develops during the course of chorea has the same significance as that developing during the course of rheumatic fever and other diseases.

Treatment.—The main objects of treatment are :

1. To obtain rest (mental and physical).
2. To improve nutrition.
3. To give such drugs as may be thought to have a special influence on the disorder.

1. **Rest.**—In cases of any severity the child should be kept in bed. In others the time should be spent quietly, free from all excitement, and when possible it is desirable to avoid the society of other children.

2. **Nutrition** must be improved by a diet containing plenty of milk; preparations of cod-liver oil, iron, phosphates, and other similar tonics are very useful.

3. **Drugs.**—The fact that so many drugs are recommended for the treatment of chorea is strong evidence that no single one can be depended upon to cure it.

The drug that has gained most popularity is arsenic, either given in rapidly increasing doses over a short period of time, or in smaller amounts over a longer one. In the first instance, where the dose begins with 2 or 3 minims of liquor arsenicalis and is, perhaps, quickly raised to 8 or 10 minims, careful observation must be kept for the appearance of any symptoms of acute arsenical poisoning; while in giving the drug in smaller doses over a longer period of time there is always a danger of producing peripheral neuritis. As in other circumstances, arsenical preparations should be well diluted and given after meals.

The benefit to be obtained from this form of treatment is by no means assured, and it is now generally agreed that arsenic has no specific action on the course of the disease.

Salicylates and aspirin are extolled by some, but, in spite of the close relationship of chorea to rheumatism, there is no consensus of opinion in favour of these drugs.

Phenacetin, antipyrin, and chloretone are regarded as useful.

In acute cases, chloral hydrate and the bromides may be required to induce sleep.

As a general rule, rest, good food, with cod-liver oil and iron or some other suitable tonic, should form the basis of treatment, while arsenic, salicylates, phenacetin, or other drugs may be used additionally as occasion demands.

Besides treatment by drugs, the neurotic disposition of the child needs careful consideration, and the induction of happiness and contentment often materially assists the cure.

Leonard Guthrie found that suggestion combined with passive movements, the performance of voluntary movements under guidance, and, in the later stages, correction of incoördination by means of graduated exercises, are all useful adjuncts to other methods of treatment.

HUNTINGTON'S CHOREA

This is an hereditary form of chorea, and bears no relation whatever to rheumatic chorea. It can often be traced through several generations; both sexes suffer, but it is said never to skip a generation, so that once the tendency is broken it does not reappear.

The symptoms usually begin after middle age, and consist of choreic movements and ataxy together with mental degeneration. The movements cease during rest, but are otherwise very persistent; they are slower than those of rheumatic chorea.

Ataxy is a prominent feature, so that there is often a staggering gait like that of a drunken man.

The mental changes are chiefly characterized by progressive enfeeblement.

The disease often lasts a number of years. No adequate treatment is known.

SENILE CHOREA

Occasionally in late middle life cases occur which resemble in many respects those of Huntington's chorea, but in these senile cases no other members of the family suffer. The prognosis is not good.

ELECTRICAL CHOREA

Electrical chorea is the term applied to a rare condition in which sudden movements like those from an electric shock are associated with wasting of muscles and pyrexia. This disease, which progresses to a fatal termination, was first described by Dubini, and appears to occur in Italy only. The cause has been ascribed to some infective process.

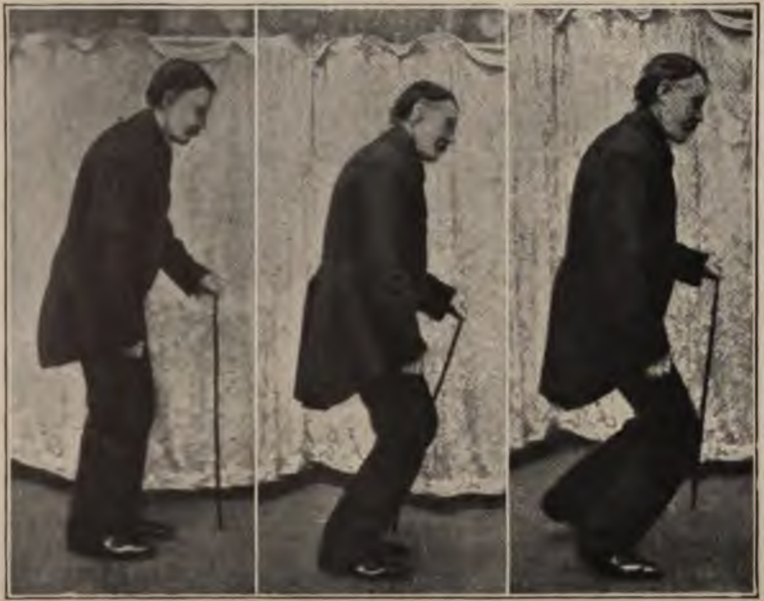

PLATE 23.—Successive stages of stumbling movements in a peculiar gait due to a coordinated tic.

CHAPTER XLIX

THE TICS

THE tics are spasmodic movements of psychical origin which are repeated time after time, without any definite cause or object, and are, for the most part, varieties of the compulsion neuroses (*see* p. 498).

They usually represent voluntary movements which would be useful in their proper place, and their origin can often be traced to the repetition of movements which were at one time performed with a definite purpose. In this way persistent blinking may result from errors of refraction or injury to the eye; sniffing, from nasal affection; and shrugging the shoulders, from ill-fitting clothes. Normally, all such movements should cease when the causes which evoked them are removed, but in neurotic or debilitated people they may become a habit and persist indefinitely. Several different movements may be out of hand at the same time, such as blinking the eyes and twitching the mouth muscles.

These tics must not be confused with the spasms which sometimes accompany severe attacks of trigeminal neuralgia, and which have received the name of tic douloureux.

The varieties of tics are innumerable. When the movements are confined to a small group of muscles, e.g. in blinking or sniffing, they are often called *simple tics* or *habit spasms*, while the more complex ones are designated as *coördinate tics* (Plate 23), though all true tics are, as a matter of fact, coördinate movements.

There are also *mental tics*, in which an idea or a tune constantly repeats itself, though here, again, it must be understood that there is a psychical element present in the production of all tics. *Tics of speech* also occur and may coexist with impulsive movements of the body; in some of these the patient repeats some words which he has just heard spoken (*echolalia*), and in others blasphemous or obscene expressions are habitually involuntarily uttered (*coprolalia*). *Latah*, or the jumping disease, found especially among Malays, is probably a complicated form of tic.

Etiology.—Allusion has already been made to the origin of tics from movements which were at first performed with a definite object. Heredity and a neurotic temperament are both important conditions, as also is mimicry. While tics of slight severity often arise from overwork or general debility, and disappear again as the health improves, many are very persistent, and depend for their origin on disturbances of the mental equilibrium as described in connection with the psychoneuroses (p. 479).

Character of the movements.—The movements constituting the tics usually consist of short, sharp spasms, which may be executed with great rapidity, and which have been compared to the movements from an electric shock; or they may be more deliberately performed with a rhythm that seems quite voluntary, while occasionally they assume a tonic character. The rapid, lightning-like jerks are generally well seen in the facial tics.

The performance of a tic is usually preceded by a desire to make the movement, and followed by a sense of annoyance at having made it, but after a time the movement may become so habitual that it takes place unconsciously.

The spasms are apt to occur in bouts, with intermissions of varying length. Any attempt to control the movements involves great effort, which can only be sustained a very short time, and is usually followed by a specially severe attack. The tics, as a general rule, disappear during sleep.

Diagnosis.—In children the movements are liable to be mistaken for those of *chorea*.

The distinction can be made by noting that—

1. The same movement or movements are constantly repeated in tics, whereas in chorea the movements are irregular and various.

2. The movements in tics have a more distinct, purposeful character, though they may have no adequate cause and bear no definite result. Those of chorea are irregular and in no way suggestive of purpose.

3. In the tics there are definite bouts, with intervals of complete freedom. In chorea there are exacerbations, but the intervals are occupied by movements, even though slight, so that there is not the complete intermission that is present in the tics.

From reflex spasms.—It is very important to distinguish between spasms due to reflex irritation and those of cortical origin, which constitute the tics. Thus, as already mentioned, tic douloureux, a reflex spasm arising from the irritability of the 5th nerve in severe neuralgia, has nothing of the characters of the true tics.

Reflex spasms of the sterno-mastoid, trapezius, and other muscles of the neck may all give rise to *torticollis*, and must be carefully distinguished from tics of cortical origin. A physiological as opposed to an anatomical distribution of the movements will often be of some assistance. For instance, a coördinated action between different groups of muscles, such as the sterno-mastoid on the one side and the deep rotators of the neck on the other, is much more suggestive of cortical origin than of some local nerve trouble that has by coincidence happened to irritate two widely separated muscles which physiologically tend to act together.

Prognosis.—The outlook varies considerably for different cases. In children, simple tics often entirely disappear, but in adults the result is not so hopeful.

Those which have arisen during a period of special strain are more likely to disappear in favourable circumstances than those that have come on insidiously during the ordinary daily life. In many the movements diminish with improvement in the general health, but are apt to show themselves again very readily during worry or excitement.

Treatment.—Any source of peripheral irritation which causes or aggravates the spasm must be removed; next, the general health must be attended to. Electricity is useful, but needs care. Where the movement tends to assume a tonic character, as in some of the cases of torticollis, faradism of the opposite muscles is sometimes beneficial, while galvanism may be applied to those at fault.

Carefully graduated exercises, whereby the patient learns to arrest some movements and to make others more regular, are often very useful, and may be practised in front of a looking-glass. A course of psychotherapeutics, which may be combined with massage and general exercises, will often be found beneficial in selected cases.

The treatment of some forms of localized tics by surgical measures is tempting, but must always be approached with great caution. Only very exceptionally are these measures appropriate; the recollection of this will often avert the disappointment of finding the spasm extending to other muscles after the nerves to those originally affected have been cut. On the other hand, the spasms of reflex origin may often be more favourable for surgical intervention.

Nodding Spasms (Spasmus Nutans)

Rhythmical movements of the head, either of a nodding or of a side-to-side character, associated with a lateral or rotatory nystagmus, are frequently met with in infants. The origin is not clear, but the usual associated conditions are debility, teething, or rickets.

The prognosis is good, and in most of the cases the abnormal movements, both of head and of eyes, cease as the child grows older. Cod-liver oil and other tonics are useful in treatment.

CHAPTER L

MYOCLONUS; MYOTONIA CONGENITA; FAMILY PERIODIC PARALYSIS

MYOCLONUS (PARAMYOCLONUS)

Myoclonus is characterized by rapid, sudden contractions of muscles resembling those obtained by an electric shock.

Etiology.—Nothing definite is known about its causation beyond the fact that debility and ill-health seem often to have preceded it. It sometimes occurs in several members of the same family, and it is apt to be associated with epilepsy.

Symptoms.—The characteristic symptoms are repeated, sudden, electric-shock-like contractions of muscles, chiefly of the limbs, less often of the trunk, and least often of the face.

The contractions are usually bilateral, and occur on both sides either at the same time or immediately after one another. If they are sufficiently strong and extensive the whole limb moves, or in severe cases the patient may be thrown down. The spasms usually cease during sleep, and are often ameliorated during voluntary movements. The reflexes are generally exaggerated, and the nutrition of the muscles remains good.

The seat of the disease is uncertain, but from its occasional association with epilepsy the cerebral cortex seems to be the most likely place.

Diagnosis.—The movements differ from those of *rheumatic chorea* in their suddenness, persistence, and distribution, while there are not the wasting and general symptoms met with in electrical chorea. The *tics* may be confused with myoclonus, but they are more purposive and more likely to be one-sided.

Prognosis.—There is no certainty about the course of these cases. In most of them the movements persist, but in a few they cease spontaneously or as the result of treatment.

Treatment.—In the absence of any discoverable source of irritation, the treatment must be conducted on general hygienic principles. If the spasms are very violent, sedatives, such as hyoscyamus, may be needed, and if epilepsy is present the bromides should be given.

MYOTONIA CONGENITA (THOMSEN'S DISEASE)

Etiology.—This disease appears to be due to some deficiency in development, and the symptoms generally appear during childhood. It is essentially a family disease, and is frequently handed on through several generations.

Pathology. — Some hypertrophy of the muscles often occurs, but there do not appear to be any changes in the central nervous system. It seems possible that this is one of the conditions which may be at any rate partially explained by a want of proportion in or modification of the two main constituents of muscle fibre (p. 42), so that the power of the substance causing tonic contractions becomes the dominating feature.

Symptoms.—This disease is characterized by the occurrence of spasms at the beginning of a voluntary movement, which gradually pass off with the persistence of the movement.

On attempting to walk after resting, the legs are stiff and can at first only be moved with difficulty; when the movement is once established they become supple, and remain so until after another interval of rest.

The difficulty in starting a movement is well seen by asking the patient to open and shut his eyes or his hands.

The muscles are usually well developed and even hypertrophied, though there is often some degree of weakness to be detected.

Electrical reactions.—The reaction of the muscles is different from normal to both the faradic and constant currents. With the faradic current a longer, more tetanic contraction than usual is obtained. To the galvanic current the muscles are unduly excitable, and the strength of the contraction obtained by the closure of the anode (ACC) approaches or becomes equal to that of the kathode (KCC). Direct percussion of the muscles also causes a slow contraction.

Prognosis and treatment.—The disease does not shorten life.

The only form of treatment which appears to produce any benefit is that of gymnastic exercises.

FAMILY PERIODIC PARALYSIS

This disease, as the name implies, runs in families and is also transmitted from one generation to another. It occurs in both sexes.

Etiology.—The family and hereditary distribution is suggestive of some developmental disorder, while the periodic nature of the attacks seems to point to the likelihood of a temporary toxæmia.

Excessive fatigue, specific fevers, constipation, and indigestion are among the conditions that are thought to dispose to the onset of the attacks.

Symptoms. — The characteristic symptoms are temporary paralysis with loss of reflexes and electrical reactions.

The attacks occur from time to time after intervals of various lengths. The onset is generally during the night, the patient waking up to find that he cannot move his limbs. Observation has shown that power is generally lost in the legs first, and later in the arms and trunk. The muscles supplied by the cranial nerves are occasionally weakened, in which case there may be symptoms of facial and bulbar paralysis.

The paralysis is of a flaccid character, and the deep reflexes and electrical reactions in the paralysed muscles are diminished or even lost.

The heart may be dilated and irregular in its action during the attacks.

Respiration is impaired by the paralysis extending to the trunk muscles, but it is kept up by the diaphragm, which, fortunately, appears but seldom to be seriously affected.

Sensation and consciousness are not impaired.

After some hours, power begins to return in the limbs in the reverse order to that in which they were affected.

Prognosis.—The outlook, so far as life is concerned, is usually good, but occasionally death occurs during an attack.

Treatment.—On the assumption that some poison is present, the diet should be plain and the excretory organs should be kept active. Diuretics, especially citrate of potash, have been recommended for shortening and lightening the severity of the attacks.

No constant results seem to follow the administration of drugs, but, of course, strychnine, digitalis, and others are very necessary to assist a failing heart or an enfeebled respiration, and in the case of the latter it may be necessary to give oxygen and to perform artificial respiration as well.

CHAPTER LI

OCCUPATION NEUROSES

ALL occupations which necessitate for their performance a constant repetition of exactly the same movements are liable, after a varying length of time, to become difficult or impossible to carry out. As a general rule, the more delicate and accurate the repeated movements require to be, the more likely is the power of performing them to be lost, especially if constant concentration and mental strain be required at the same time; and, further, individuals who are naturally of nervous temperament are probably the most likely to suffer this loss of power.

Among those especially liable to be troubled in this way are writers, piano and violin players, typists, and telegraphists; the affection is also met with in workers in many other occupations, such as seamstresses, cigarette makers, and dairymaids. One of the most interesting cases that have come under the writer's notice was that of a man whose chief business was to sharpen razors, and who was unable to carry the blade smoothly backwards and forwards on the stone on account of sudden little spasms of the arm which quite spoilt the accuracy of the movement.

Writer's cramp, which is one of the most familiar of occupation neuroses, occurs mostly in those who have to write a stiff, copy-book hand for many hours a day. It is distinctly connected with a prolonged cramped movement, for it does not seem to occur in those who write a free hand from the shoulder. Clerks whose chief business it is to be continually copying long documents are among the most common sufferers.

Symptoms.—The symptoms are mainly those of cramp in the small muscles of the hand. After writing for a longer or shorter time, according to the severity of the case, the fingers become stiff and involuntarily separate themselves, so that it is impossible to hold the pen, which is spasmodically dragged across the paper or even shot out of the hand; in some cases there is, every now and then, a strong flexor spasm.

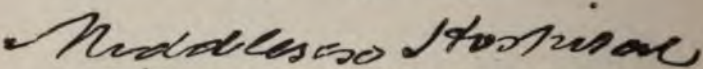
Middlesex Hospital

Fig. 113.—Specimen of handwriting in writer's cramp.

Pain is a variable symptom, but there is often discomfort, with tingling and aching sensations. In some cases weakness is a more prominent feature than spasm.

At first the patient finds he cannot write so long or so quickly as he could formerly, and then the difficulty becomes greater and greater until at last his occupation is gone.

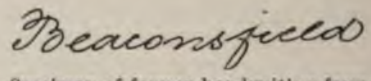
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Fig. 114.—Specimen of former handwriting from the same case as in Fig. 113.

The accompanying figures (Figs. 113, 114) show how the style of writing may be affected.

Most movements other than those concerned in writing can often be carried out with care, but occasionally some of the finer ones suffer a little, although usually not to anything like the same degree.

The electrical reactions are often unchanged. In severe cases the muscles are said to be over-excitable; more rarely the reactions are diminished.

Seat of the lesion.—The cerebral cortex, the peripheral nerves, and the muscles themselves have all been regarded as at fault, but it is difficult to see how a change in a peripheral nerve can affect one set of movements to the exclusion of others. Nor is there any good evidence that the fault lies in the muscles.

The hypothesis most in harmony with the facts is that the trouble is of cerebral origin and due presumably to incoördination following exhaustion of the particular combination of nerve cells from which the movement originates. "The education of centres which may be widely separated from each other for the performance of any delicate movement is mainly accomplished by lessening the lines of resistance between them, so that the movement, which was at first produced by a considerable mental effort, is at last executed almost unconsciously. If, therefore, through prolonged excitation, this lessened resistance be carried too far, there is an increase and irregular discharge of nerve energy, which gives rise to spasm and disordered movement. According to this view, the muscular weakness is explained by an impairment of nutrition accompanying that of function, and the diminished faradic excitability by the nutritional disturbance descending the motor nerves."—Gay (quoted by Osler).

In other instances the loss of movement is not so strictly limited, and pain along the course of a nerve, with perhaps some slight evidences of a muscular wasting and changes in electrical reaction, seems to point to the possibility that the trouble is connected with the peripheral nerves.

Diagnosis.—When a patient complains of difficulty in writing, it is necessary to exclude other possible causes before concluding that it is a case of writer's cramp.

1. Examine the hand and wrist for any local trouble such as teno-synovitis.

2. Ascertain if there are any signs of localized neuritis, as shown by weakness in all movements, pain and wasting, or of multiple neuritis with a tendency towards a symmetrical distribution.

3. Remember that tremor from any cause will spoil the writing; and bear in mind, among other things, the possibility of commencing paralysis agitans, disseminated sclerosis, and general paralysis of the insane.

Prognosis.—The prognosis is not good, so far as the possibility of recovering power to perform the particular movement is concerned. It is only seldom indeed that anyone who develops an occupation neurosis to an advanced degree is ever able to earn his living again by that particular movement, but by taking trouble and assisting with the other hand the patient is often able to continue for a considerable time before having to give up. Cases in which the lesion seems to be in the periphery are probably more favourable than those of cortical origin.

Treatment.—Complete rest from the particular occupation at the very first sign of the trouble gives the best chance of ultimate recovery. Occupations involving other and coarser movements may be continued, provided that care is taken to avoid tiring the affected limb. Locally, massage, hydropathic treatment, and galvanism are all beneficial. The faradic current should generally be avoided.

If the pain and spasm persist when the occupation has been left off, such drugs as bromides, phenacetin, and antipyrin may be given, but the main effort in general treatment should usually be towards improving the health by rest and a nutritious diet with plenty of milk and, if needful, cod-liver oil. Various devices are usually adopted by the sufferers to put off the evil day of relinquishing their work. In writer's cramp the patient finds he can get along better for a time by holding the pen between the first and second fingers, and from this he takes to a very thick penholder, which he grasps with the whole hand. Such devices, however, can only temporarily hide an irremediable deterioration.

CHAPTER LII

PSYCHONEUROSES

GENERAL INTRODUCTION

THE Psychoneuroses comprise a group of cases in which the symptoms represent some degree of failure of the individual to adapt himself adequately to the realities of his daily life. Among the principal varieties are Hysteria, Compulsion Neuroses, Neurasthenia, and Anxiety Neuroses.

A nervous breakdown is, generally speaking, due to a disturbance of equilibrium between the adaptability of the individual and the complexity of his surroundings. In one case the power of adaptation may be abnormally weak, while in another the difficulties to be overcome may be unusually great. It is on the relative strength of these two factors that the resulting type of nervous reaction depends, as evidenced by the strength or weakness with which people respond to the ordinary difficulties of daily life. That defective power of adaptation, which may be either inherited or acquired, is of great importance has been shown by consideration of " shell shock " cases, the histories of which frequently revealed a previous breakdown owing to inability to meet the requirements of even the ordinary conditions of civil life.

The symptoms are caused by a disturbance of the contents of the mind, which contents, according to modern conceptions of psychology, are to be considered under the aspects of the " conscious," the " subconscious," and the " unconscious " mind.

The " conscious mind " represents all those thoughts which are known to the person at any given time.

The " subconscious (preconscious or foreconscious) mind " is concerned with ideas which, though not in the field of consciousness at a given moment, can easily be called up by the processes of association of ideas.

The " unconscious mind " contains memories and ideas which can only be called into consciousness with great difficulty, usually by the aid of such methods as hypnotism or the free association of psychoanalysis.

According to this conception, there is free interchange always taking place between the ideas of the conscious and the subconscious levels, but very little, if any, between those of the conscious and the unconscious levels; and it is in the unconscious plane that memories of a painful nature and ideas representing such broad primitive desires as self-preservation and race-preservation are repressed when full gratification of such desires is incompatible with recognized social conduct.

On this hypothesis, symptoms of " nervousness " may be brought about mainly in two ways :—

(1) By the reproduction, through processes of mental association, of the emotional state corresponding to a painful memory, the nature of the latter often being unrecognized by the patient.

(2) By a mental conflict resulting from the desire to attain ends which may be in a large degree incompatible with the conventions of ordinary life.

A simple example of the first type is given by Rows,* of a man who could ride in a bus or train without discomfort but who fell into a state of terror if he entered a tram. On examination of his mental state the disability was found to be caused by the sound of the trolly wheel on the wire, which was unconsciously associated in his mind with the sound of an approaching shell that had broken into his dug-out, with resulting injury to himself and loss of life to some of his companions.

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* Morison Lectures, 1920. *Edin. Med. Journ.*

In the second group, if the conflict between the suppressed desires and their possibilities of attainment cannot be settled satisfactorily by working off the energy in a useful manner, i.e. in the activities of everyday life, the result may be the formation of some kind of neurosis which may take various forms, e.g. an anxiety neurosis, a compulsion neurosis such as a tic, or a so-called conversion neurosis, in which peace of mind is sought at the expense of physical disability, as occurs in cases of hysterical paralysis.

On the bodily side any conditions which impair the nutrition of the nervous system, as fatigue (mental or physical), injury, toxæmias, arterio-sclerosis, will facilitate the development of changes in the mental state.

Particular mention must also be made of the internal secretions (the endocrinic system), which recent researches have shown to have an important influence on nervous metabolism through the autonomic system (*see* p. 48).

When mental balance is upset in one of these ways a state of "nervousness" and "apprehensiveness" is apt to arise, and upon this state further mental and bodily symptoms may develop according to the type of the patient and the surroundings in which he is placed.

The ease or otherwise with which such a "nervous" state is brought about depends upon the degree of stability of the individual in relation to the intensity of the debilitating conditions by which he is affected.

There are some persons whose nervous systems are so constituted that they quickly feel any undue stress, and are unable to adjust themselves adequately even to the average circumstances of daily life. Such people are in a more or less chronic condition of nervousness, which varies according to their state of general health and to the surroundings in which they happen to be situated. On the other hand, those who possess nervous systems of average or greater than average stability will only become nervous from some unusual stress of mind or body.

Assuming the nervous symptoms to begin as the result of some shock or mental conflict as suggested above, the sequence of events may be broadly indicated as follows :—

Mental shock or conflict.

Emotion.

Bodily disturbance, mainly through autonomic system.

(*a*) Mental disturbance, feeling of apprehension, and general "nervousness."

(*b*) Possible further development of more definite mental symptoms, e.g. obsessions, general lack of perspective, suspicion, hypochondriasis, or delusions.

The more definite developments enable further classification to be made, and among the principal states thus recognized are those known as Neurasthenia, Anxiety Neuroses, Compulsion Neuroses and Hysteria. It must, however, be distinctly understood that no very definite line can be drawn between these different varieties, for frequently there are many symptoms common to all, and the classification is to be justified on grounds of convenience rather than of reality.

HYSTERIA

Under the term hysteria are included a variety of well-recognized symptoms which appear to depend essentially upon a disorder of mind.

It is obvious that symptoms depending on such a cause cannot have any sharply defined boundaries, for many organic diseases are naturally often preceded or accompanied by similar symptoms due to changes in the irritability and conductivity of the nervous system before any signs of organic destruction can be detected, as also are many of the more severe "functional diseases," among which are included some of the forms of insanity.

In the absence of any structural change, the exact nature of the changes, if any, underlying hysteria must still be regarded as unknown. Since the time when the symptoms were thought to be caused by movements of the uterus many hypotheses have been suggested, some of which place the disease on a physical and others on a mental basis. Among the more important of the views put forward in recent times are those of Janet, Babinski, and Freud.

Janet's view is based upon the phenomena of dissociation of consciousness. "Hysteria," he says, "is a form of mental depression, characterized by the retraction of the field of personal consciousness and a tendency to the dissociation and emancipation of the systems of ideas and functions that constitute personality." *

According to Babinski, the characteristic of an hysterical symptom is that it can be produced by suggestion and made to disappear by persuasion, a condition of things to which he has given the name *pithiatism* (πειθώ, persuasion, ἰατός, curable). On this basis he considers hysteria to be a pathological condition showing itself by a set of primary symptoms which, in certain subjects, it is possible to reproduce exactly by suggestion, and which can be made to disappear again solely by persuasion. In this group of primary symptoms, which have in common the characteristics of being reproducible by suggestion and removable by persuasion, Babinski places convulsions, paralyses, contractures, tremors, rhythmical or irregular choreiform movements, and disturbances of sensation, respiration, and phonation.

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* "Major Symptoms of Hysteria."

There is, however, a group of secondary symptoms which are not directly dependent on suggestion for their production, including exaggeration of cutaneous reflexes, tachycardia, erythemata, and excessive secretion of the skin or the intestine.

Freud considers that hysteria is the result of painful experiences (mental traumata), the memories of which, through their unpleasantness to the patient, have become dissociated from the conscious part of the mind and repressed into that of the unconscious. The natural end of such painful mental experiences is neutralization by suitable action or gradual dimming of their effects by time; but when for any reason the impressions are not thus neutralized, the memory is apt to be buried only and not destroyed, and is able then in certain circumstances to resume its activity and to influence the feelings and activities of the patient by clashing with his general ideas of behaviour. According to these views, the hysterical symptoms (e.g. paralyses and anæsthesias) represent manifestations of certain complexes that have become dissociated or "split off" from consciousness and form the type known as *conversion hysteria,* in which the mental trouble is got rid of at the expense of physical disability. In another form, spoken of as *anxiety hysteria,* the principal symptoms are apprehension, phobias and hysterical attacks similar to those occurring in the group of "anxiety neuroses." Space will not permit Freud's theories to be dealt with in further detail; for fuller information the reader should consult special books on the subject.

Etiology.—Hysteria occurs more frequently in females than in males, although the latter are by no means exempt; quite young children may also be subjects of the disease. It is common for the symptoms to appear about the time of puberty or soon afterwards. Heredity is an important factor, as also are the natural temperaments of different nationalities. The first symptoms may set in after a mental or physical shock. Lastly, it must never be forgotten that some definite organic lesion may be associated with the advent of hysterical manifestations.

Symptoms.—The symptoms are so numerous and ill-defined that it is very difficult to classify them satisfactorily, but some such grouping as the following may be found useful:

1. *Motor system*	Under-action.	Paralysis.
	Over-action.	Tremors. Spasms and contractures. Convulsions.
2. *Sensory system*	Under-action.	Anæsthesia.
	Over-action.	Pain. Hyperæsthesia. Hysterogenetic points.
3. *Special senses*	Eyes.	Spasms of ocular muscles and defects of visual power.
	Ears.	Deafness.
	Taste.	Absent or modified.
	Smell.	Absent or modified.
4. *Mental symptoms*	Impaired volition. Irritability. Depression. Exaltation. Loss of memory.	

The Motor System

UNDER-ACTION

Paralyses.—The paralyses are principally found in one limb (monoplegia), in an arm and a leg (hemiplegia), or in both legs (paraplegia), from which it is easy to see how closely they may simulate the effects of organic disease. Of these three varieties the paraplegic distribution is perhaps the most common.

The chief characteristics of these paralyses are loss of power without wasting or any electrical changes in the muscles, and frequently without definite alterations in the reflexes.

The loss of power varies in amount; it may be quite complete or only moderate in degree. Some patients can move the various segments of a limb when lying down, but are nevertheless unable to stand or walk (astasia-abasia).

Reflexes.—The *knee-jerks* are frequently increased—never absent—and there is often a "pseudo" *ankle-clonus*, which is characterized by a few short taps, generally ceasing when the foot is fully flexed. This pseudo-clonus, as it is called, is not of much value, since an incomplete clonus is often obtained in organic disease.

On the other hand, a strong and sustained clonus is greatly in favour of organic disease, though not by itself necessarily conclusive.

Plantar reflex.—The reaction of the plantar reflex is very important, for an undoubted extensor response is an indication of organic disease.

In many instances of functional disorder there are times when no plantar reflex of any kind can be obtained, and, although such cases are suggestive of functional disorder, too much stress must never be laid on a negative result, since experience shows that a plantar reflex which is either flexor or is not obtainable on one day may on another give a definite extensor reaction.

One may sum up the comparative importance of the different reactions of the plantar reflex, as far as the diagnosis of *hysterical* paralysis is concerned, as follows:—

EXTENSOR RESPONSE.—Indicates the presence of organic disease of the motor tracts.

FLEXOR RESPONSE.—Suggestive of the absence of organic disease of the upper motor tracts.

ABSENCE OF RESPONSE.—Suggestive of functional disease when taken with other symptoms, but care must be taken to see that the absence is not merely accidental or temporary, a condition which frequently occurs, e.g. from dampness or coldness of the feet.*

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* The conditions in which the plantar reflex is likely to be absent are discussed at p. 30.

Sphincters.—There may be retention of urine with overflow-incontinence, but never the true incontinence that occurs when the lumbar enlargement of the cord is damaged.

Larynx.—Paralysis of the laryngeal movements is common, and gives rise to aphonia, which often disappears as suddenly as it arises. It is always the adductor movements that are affected, so that the cords are widely separated and cannot voluntarily be brought together, although they move freely during respiration and coughing when these acts are automatically performed without the intervention of the will.

The paralyses of the larynx and, more rarely, those of the face are the only ones that are likely to occur in the distribution of the cranial nerves. The squints and ptosis which may be met with are usually due to spasms, and are referred to in the section below, headed Over-action.

General summary of the diagnosis of hysterical paralyses.—The procedure in diagnosing hysterical paralysis does not differ from that required in the diagnosis of loss of power from other causes.

The possibilities must all be carefully considered, and the diagnosis of hysteria only arrived at after rigid exclusion of everything else. Assuming the patient to have *lost power in the legs,* the following questions must be asked:

1. *Is the lesion situated in the upper or lower neurone?*—(Upper, on account of increased or normal tendon reflexes, absence of wasting, and electrical changes in the muscles, with possibly some active contractures.)

2. *Is the lesion in the spinal cord?*—Consider the reactions of the reflexes (knee-jerks, tendo-Achillis jerks, ankle-clonus, and plantar reflex). If the lesion is due to an anatomical change in the lateral columns, these should all behave consistently. An organic lesion of the lateral columns which produces exaggeration of the knee-jerks should, in the ordinary course of things, also give rise to an extensor response of the plantar reflex, and, in all

probability, to an ankle-clonus. If, therefore, the signs are contradictory, as they are when an exaggerated knee-jerk exists with a flexor or an absent plantar response, suspicion may be aroused as to the likelihood of an anatomical lesion being present.

On the other hand, the absence of knee-jerks entirely negatives hysteria, as also does a typical extensor response of the plantar reflex.

3. *What is the condition of the sphincters?*—Retention of urine is not conclusive evidence of either functional or organic disease, but true incontinence is indicative of organic change.

4. *If anæsthesia is present, what is its distribution?*—The anæsthesia of organic disease will be distributed according to anatomical rules. In hysteria the intensity and distribution of the anæsthesia may vary from time to time, and usually does not correspond in any way to the distribution that would follow an organic lesion.

Additional considerations. Gait.—The gait in hysterical paralysis assumes a variety of forms, some of which are obviously too complicated to depend upon organic lesions. In hysterical hemiplegia the affected leg is dragged along with the upper surface of the toes on the ground, instead of being stiffly swung round from the hip-joint as it is in the organic spastic cases. The hysterical paraplegics generally shuffle along without being able to raise the soles of the feet from the ground. A further test devised by Babinski for distinguishing between hemiplegia of organic and hysterical origin is described under Hemiplegia, p. 397.

OVER-ACTION

Tremors.—The intensity of hysterical tremors may vary from the finest to the coarsest movements, and, like other hysterical manifestations, they are apt to differ very widely from time to time. They often closely simulate the tremors of organic disease, but can generally be distinguished from them by the absence of other symptoms which would naturally be associated with the tremor, and by the failure of the tremor to behave continuously like those known to be associated with organic disease.

Spasms.—Localized spasm of muscles is of common occurrence, and may be observed in connection with muscles supplied by both cranial and spinal nerves. (Fig. 115.)

Of the muscles supplied by cranial nerves, the most commonly affected are those of the eye, and more rarely those of the tongue (Fig. 116) and the jaw.

Eyes.—Spasm of the muscles of accommodation is frequent, as also are spasmodic ptosis and convergent strabismus. The ptosis, which at first sight may appear to be a paralytic manifestation, is due to spasm of the orbiculares palpebrarum, and is unaccompanied by any compensatory contraction of the occipito-frontalis, such as occurs with the paralytic form of ptosis. In a few rare instances, hysterical ptosis due to relaxation of the levator palpebræ has been recorded.

Hysterical rigidity and contractures of the limbs (Fig. 117) are often exceedingly tense, and any endeavour to overcome them by force only increases the opposition, nor does an attempt to approximate the points of insertion of the contracted muscles aid their relaxation, as it does in the cases of organic origin. Relaxation is, however, usually effected during sleep, and generally by the aid of an anæsthetic.

Contractures are especially common in the feet, knees, and hips, and may be accompanied by great pain.

Catalepsy, during which the limbs remain for a long time in any position in which they are placed, sometimes occurs.

Spasms of respiratory muscles.—Irregular spasmodic action of the muscles of respiration is associated with hysterical hiccough, yawning, cough, and globus. Hysterical dyspnœa may also be present.

Spasms of the œsophagus with difficulty in swallowing may suggest the presence of a stricture of organic origin.

Convulsions.—Hysterical convulsions, which in many respects bear a close resemblance to epileptic fits, may occur. They are usually ushered in by some hysterical sensation, as the globus hystericus, flatulence, or palpitation. Generalized tonic spasms then occur, and the patient falls or slides to the ground, but

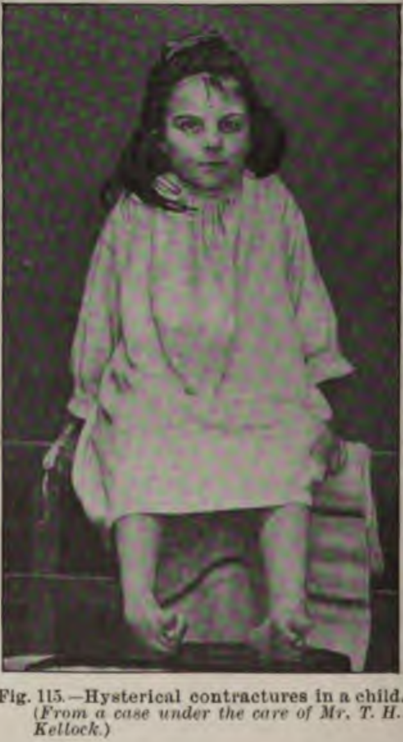

Fig. 115.—Hysterical contractures in a child. *(From a case under the care of Mr. T. H. Kellock.)*

seldom with the sudden helplessness that is so characteristic of epilepsy. The tonic stage is succeeded by clonic movements which are often very wild and uncontrolled, and in severe cases opisthotonos and different elaborate postures of the body are assumed. After a variable time, usually much longer than in epilepsy, the movements cease, and the normal mental condition gradually returns. The main diagnostic points between these fits and epilepsy are mentioned in the chapter on Epilepsy (p. 442).

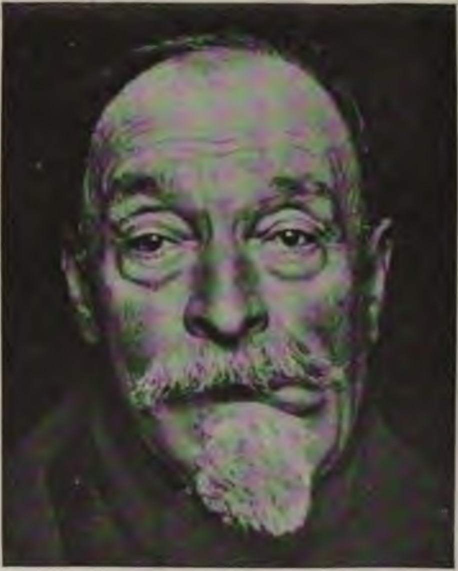

Fig. 116.—Hysterical spasm of the tongue.
(*Pierre Marie.*)

Lastly, among the infinite variety of spasms and movements that are met with may be mentioned the dancing movements known as *saltatory chorea.*

Sensory Symptoms

Anæsthesia.—Anæsthesia is the symptom most constantly present in hysteria. The variability of its distribution and intensity is well known, but the most frequent distribution is over one half of the body, the distal portion of a limb, or a segment of a limb (Figs. 118, 119, 120). It may also be present in irregular patches.

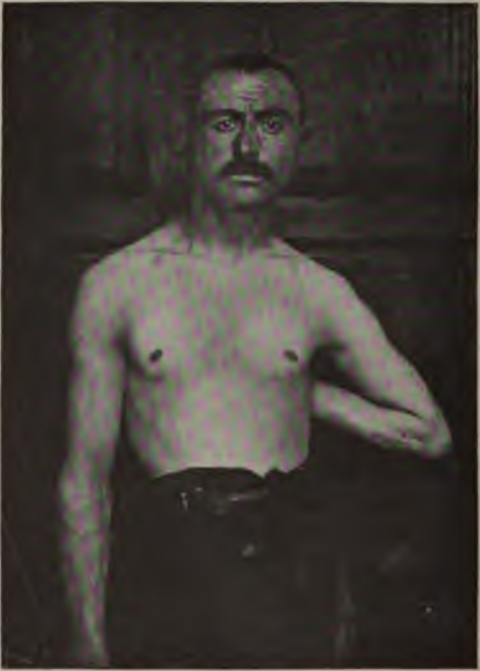

Fig. 117.—Hysterical spasm of the arm. (*Pierre Marie.*)

In **hemianæsthesia** the loss may be complete for all kinds of sensation, or it may be partial, or there may be loss for one kind of sensation and not for another. The sensory loss usually extends to the middle line of the body and affects mucous membranes as well as skin. In some instances the sensations of the deeper structures, e.g. muscles, bones, joints, and ligaments, are lost; and in such cases, if the eyes are closed, the patient does not recognize the position or even the presence of the limb.

The **special senses** are also often defective at the same time. The anæsthesia may disappear during sleep. Sometimes a touch on one limb is felt on the opposite limb (allocheiria).

Hyperæsthesia.—In some instances sensation is abnormally acute over circumscribed areas, and pain and tenderness may be prominent symptoms. Pains may simulate those of rheumatism, migraine, and angina; headaches are common, and may be severe enough to suggest the presence of a tumour. Tender points (hysterogenetic spots), pressure on which may start a convulsion, often exist over various vertebræ, or below the mammæ, and in women over the ovarian regions.

Visual symptoms.—Allusion has already been made to the spasms of the ocular muscles. The fields of vision are frequently contracted in a manner characteristic of the disease: there is constriction of both fields, but more so of one than the other, the constriction being greatest on the side of the anæsthesia. This condition is known as *crossed amblyopia.* The chart of the field of vision in these cases often has a spiral or corkscrew appearance, owing to the field becoming progressively smaller as it is being taken.

Hemianopia from hysteria, if it occurs at all, is so rare that it need not be considered. Recognition of colours is sometimes modified so that everything appears a uniform grey (*achromatopsia*). Short of this the field for red is the last to be lost, whereas in organic disease it usually fails first. Complete hysterical blindness is rare.

Deafness and loss of *taste* may also be present on the same side as the hemianæsthesia. When loss of *smell* occurs it is generally bilateral.

Gastric symptoms.—There may be loss of sensation

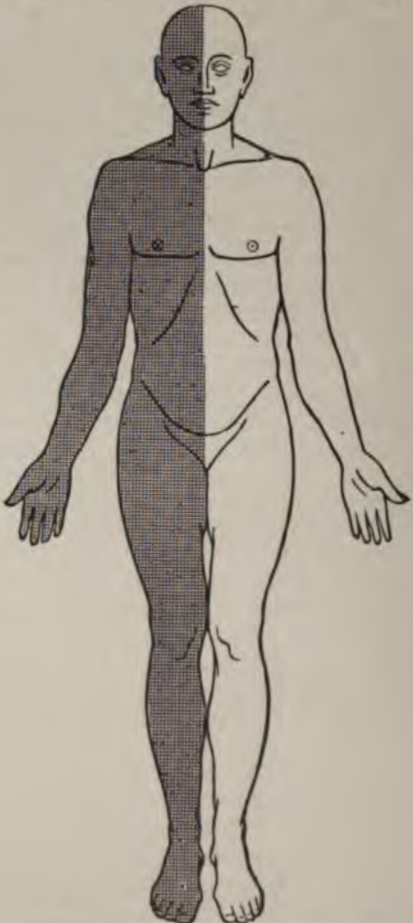

Fig. 118.—Hysterical hemianæsthesia.

of hunger, or there may be undue craving for various foods. Sometimes there is vomiting. Obstinate constipation may be present.

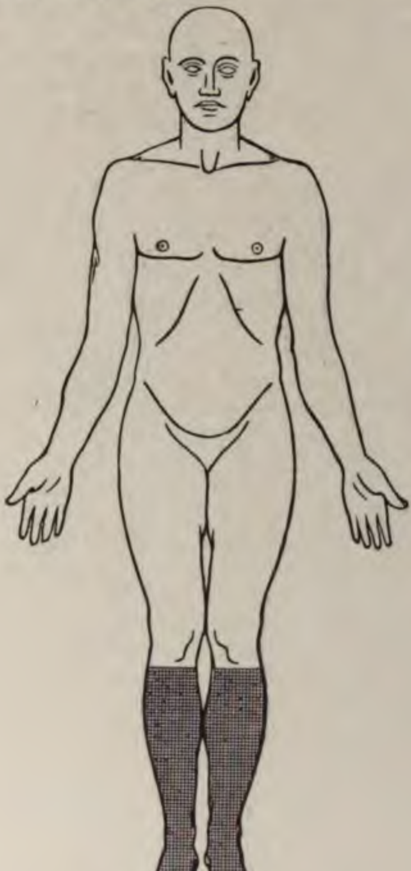

Fig. 119.—A type of hysterical anæsthesia.

Vaso-motor disorders, such as flushing, erythematous eruptions, localized œdema, and even cutaneous hæmorrhages, may occur. Dermographism, the condition in

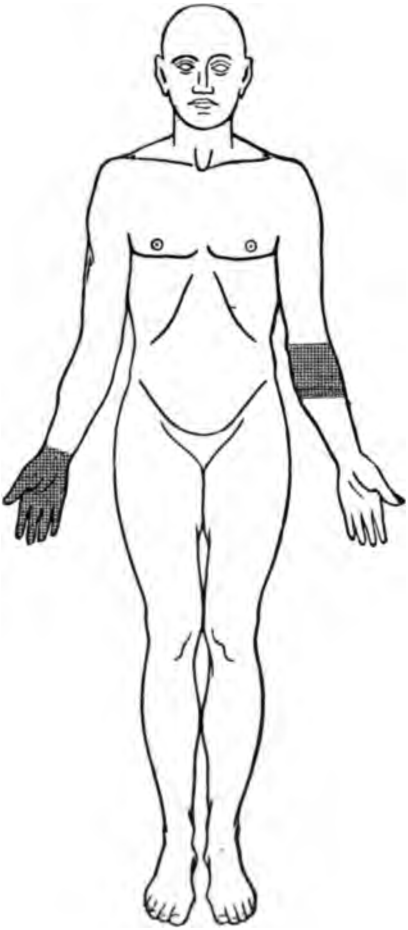

Fig. 120.—Types of hysterical anæsthesia.

which wheals are readily brought out by drawing the finger-nail across the skin, as in the case of those suffering from urticaria, may also be present.

Temperature.—The temperatures of extraordinary height that are occasionally recorded in hysteria are probably of artificial production. A genuine rise of temperature over any length of time should arouse suspicions of the existence of underlying organic disease.

MENTAL SYMPTOMS

Capriciousness, rapid change of moods, unreasonable likes and dislikes, are among the prominent mental symptoms of hysteria. There is a general exaltation or depression of the emotional side of the mind, with weakening of the power of judgment. In short, the nervous system is unstable, and the most various reactions may result. Craving for sympathy sometimes exists to such a degree that self-mutilation is practised in order to gain it. In severe cases, cataleptic conditions may be met with. Amnesias, i.e. the occurrence of blanks in the continuity of memory, are met with, and "double personalities" also occasionally occur.

Diagnosis.—Hysteria may simulate the effects of almost any organic disease, and the diagnosis in some cases may be very difficult, for beyond the decision that this or that symptom is "functional," one has to consider whether there is a background of organic disease upon which the hysterical symptoms are superimposed. The only way of avoiding mistakes is to search thoroughly for evidence of organic disease in the past history and present condition of the patient. The chief signs of organic disease which should be looked for are paresis of ocular muscles, fixed pupils, nystagmus, hemianopia, optic neuritis and atrophy, loss of tendon reflexes, extensor response of the plantar reflex, a true ankle-clonus, and incontinence of urine. It may be said that evidence can be adduced to show that some of these signs can appear in hysteria, but the occurrence of, at any rate, most of them is exceedingly rare, and for practical purposes it is safe, whenever the nature of the case is doubtful, to regard them as indications of the presence of organic disease.

Stress has elsewhere been laid upon the danger of confusing disseminated sclerosis with hysteria (p. 216), and the diagnosis of the hysterical convulsions from epilepsy has also been discussed (p. 442).

Prognosis.—The prognosis is, on the whole, good, but there is in many instances a tendency to relapse. Some cases recover spontaneously, or respond rapidly to suitable treatment, but others may take months or even years before they are cured. All the symptoms may disappear quite suddenly.

Treatment.—Mild cases of hysteria often recover with improvement in general health and change of surroundings, especially if the latter is accompanied by effective moral control. In others some course of special treatment is indicated, details of which will be found at p. 505.

Among the drugs found useful are valerian and bromides, while anæmia or general debility may be treated with tonics such as iron and arsenic.

COMPULSION NEUROSES

Mental disturbances which are chiefly characterized by want of control over certain lines of thought were formerly included under the title of psychasthenia, but are now generally classed as compulsion neuroses. Among these are the different phobias, e.g. the fear of being in open spaces (agoraphobia) or in closed rooms (claustrophobia), the fear of fire or of contamination, the dread of travelling in trains, and various other obsessions, e.g. the desire to perform some useless action such as touching consecutive posts of a railing, or to repeat some meaningless words or sentences. These symptoms may occur alone, or may be associated with neurasthenia or other neurotic states.

Treatment should be conducted along the same physical and psychical lines as are described for neurasthenia and hysteria (p. 505). The general views as to the origin of these various phobias have been described at p. 479, and such methods of physical and mental therapy must be chosen as seem most suitable for each case.

NEURASTHENIA

The term neurasthenia has been largely used in a general sense to indicate almost any condition of functional nervous disturbance, but it has become more usual recently to seek to limit it to a particular group of symptoms in which abnormal sensations of fatigue are the predominant features.

Etiology.—What has been said of the etiology of the psychoneuroses in general (*see* p. 479 *et seq.*) applies to neurasthenia—that is to say, anything which produces debility of the nervous system, whether it be fatigue from work and worry, or exhaustion from severe illness, may set up a train of symptoms which causes the patient to fix his attention unduly on himself and to feel symptoms of bodily and mental fatigue as soon as he undertakes the ordinary tasks of daily life. Painful repressed memories, sexual conflicts and masturbation are regarded by some as important factors in the background.

Symptoms.—The symptoms may be divided into *mental* and *bodily*.

Mental symptoms.—Impulses reaching the brain of the neurasthenic cease to be interpreted in the relative proportions which they have previously borne to one another. Incidents of little importance are magnified, especially those connected with health. The slightest ache or pain is often the starting-point of some imaginary dreadful disease ; an unimportant lapse of memory is thought to signify commencing cerebral disorder, while in the mental background there is frequently a vague indescribable fear of impending trouble which entirely destroys all peace of mind. These symptoms may be shortly summed up by saying that the patient worries unnecessarily and loses confidence in himself. There is difficulty in concentrating the mind upon any intellectual work, and there is a fear of making mistakes in work, though as a matter of fact such mistakes are not generally made. Throughout it is a fear of what the patient *may* do and of what *may* happen rather than of what *is* done and what *does* happen.

Bodily symptoms.—On the bodily side the symptoms are most various.

Pains, crawling sensations, and other peculiarities are complained of both in head and in body. The headache is often characterized by a sense of pressure at the vertex, but at other times it is indistinguishable from the frontal, vertical, and occipital headaches due to other causes. Tenderness and " weakness " of the spine are frequently complained of. In the abdomen the common accompaniments are indigestion, with which is often associated some dilatation of the stomach, and constipation. Flatulence is often a most troublesome symptom, especially in women. Added to these are " sinking " and " fainting " feelings. The heart generally beats rather faster than normal, and the pulse-rate is easily varied by nervousness or excitement. Sexual debility may be present.

The limbs soon tire, and after walking there may be a dull pain in the lumbar region.

Attempts have been made to divide up cases of neurasthenia, according to the seat of the most prominent symptoms, into cerebral, spinal, gastric, and cardiac varieties, but such a division is too artificial to be of practical use.

Physical signs.—On examination, no signs of organic disease will be discovered. The deep reflexes of the arms and legs may be normal or exaggerated, but if they are absent it is a sign that something more than neurasthenia is present. The plantar reactions are of flexor type, or in some cases no reaction at all may be observed. There is no ankle-clonus, though a " pseudo " clonus, in which there are two or three feebly sustained clonic contractions that cease when the foot is fully flexed, may be obtained.

The limbs are feeble and generally wanting in tone, but there is no definite paralysis. There is frequently some fine tremor to be observed in the hands.

When the patient stands with the eyes closed there may be a certain amount of unsteadiness. This seems to be an exaggeration of that slight tendency to sway which most people experience if they close their eyes tightly, and it can nearly always be overcome if the patient will make an effort to control his balance. This swaying in neurasthenia, though classified under Romberg's sign, has nothing in common with true muscular incoördination such as is met with in tabes and cerebellar disease.

Examination of the sensory system does not reveal much, except where cases show hysterical phenomena, when some of the different forms of anæsthesia peculiar to the latter may be found.

As regards the eyes, the pupils are generally large, but they are equal, and contract to light and during accommodation.

The movements of the eyeball are normal, and there are no changes to be observed in the fundus. There may be a slight unsteadiness on extreme lateral fixation, but there is no real nystagmus. There is, however, often an error of refraction, to which allusion will again be made when discussing treatment.

The history reveals no symptoms that can be definitely associated with organic changes.

There will have been no double vision, though patients may complain of mistiness of sight, which must be distinguished from true diplopia; no loss of control over the sphincters will be found.

On testing the higher functions of the nervous system there may be signs of weakness of power of concentration and rapid onset of fatigue. The results of the "time discrimination" test, as described at p. 67, are frequently below the average, and it is often instructive to note the increased rapidity with which the test can be carried out as improvement takes place under treatment.

As already stated, the symptoms are largely of autonomic origin, and it is therefore disturbances of this system that should be looked for. Indications may be found in the skin by the irritability of the vaso-motor system (dermographism) and the muscles of the hair-follicles ("goose-skin"). The regularity or otherwise of the heart-beat can be chronicled by a sphygmograph, or in special cases by a polygraph. Attention should also be paid to the blood-pressure, and especially in connection with the rapidity of the adaptability of the vaso-motor system in different positions of the body. It is probably to a failure of such adaptability that giddiness is often to be ascribed, just as it is in one who is still weak from the effects of a severe illness. The oculo-cardiac reflex and respiratory arrhythmia of the pulse may be tested (p. 50).

In **traumatic neurasthenia** the symptoms are brought about by shock to the nervous system which may arise from direct injury to nerves, with the resulting pain and exhaustion, or by mental shocks produced through the special senses such as sight and hearing.

It thus happens that a direct injury or the fright and horror of a situation may both or either set up traumatic neurasthenia.

If, for example, a person is injured bodily, consequent pain and anxiety may wear down his power of nerve control, while in another case a person may have become neurasthenic from the fear of what seemed about to happen to himself or from the horror experienced at the sight of accidents to others, without having necessarily suffered any physical injury.

In some instances all the symptoms, including the more definite mental ones, arise immediately after the shock; but in other cases a general state of nervousness (the neurasthenic state) precedes more definite mental symptoms, as indicated in the table on p. 482. There is sometimes in traumatic neurasthenia a latent period between the time of the shock and the onset of symptoms.

Diagnosis.—It will be seen from what has been written above that symptoms included under neurasthenia merge into those of other forms of neurosis so that exact classification may not be possible. Similar symptoms are also in a general way apt to be present at the beginning of many other diseases, some of the principal of which it is as well to bear in mind while examining the patient.

General paralysis of the insane may begin with symptoms which closely resemble those of neurasthenia, and until some more definite signs develop it may be impossible to be sure of the diagnosis. As a rule, however, the resemblance is more apparent than real. The important point to which attention should be particularly given is the exact nature of the mental change that is present, especially as regards character and work. The general paralytic is not only unable to work so well, but he loses his skill and also his finer sentiments. The neurasthenic is over-anxious about himself, worries, and is unable to keep his attention fixed for long, but he does not show the tendency to mental degeneracy that is seen in the general paralytic. He may be over-anxious about his work and afraid of making mistakes, but, as a matter of fact, he seldom does make them; while, on the other hand, the failing mental powers of the general paralytic are often obvious to everyone but himself.

Definite failure of memory, marked alteration of reaction times, and other signs of degeneration as shown by mental tests are important points to be considered. The presence of any signs of organic disease, such as inequality of the pupils with loss of reaction to light, at once makes it evident, of course, that something more than neurasthenia is present.

Neurasthenics are often depressed, but if the depression is accompanied by suicidal tendency and delusions, then the case should no longer be regarded as one of ordinary neurasthenia, but should be looked upon as *melancholia*, so that proper nursing and supervision may be provided to ensure the best chances of recovery. The presence of definite anæsthesias, amnesias, and paralyses will help in the differentiation of *hysteria*, but often no sharp definition can be drawn. When the patient complains of being tired after walking a little way it is well to bear in mind the possibility of *myasthenia gravis*, which in its less pronounced forms may be confused with neurasthenia. The importance of differentiating *disseminated sclerosis* from functional disease is insisted on elsewhere (p. 217).

Treatment.—The general principles of treatment are those described at p. 505, with such modifications as are required for any particular case.

ANXIETY NEUROSES

The symptoms of this form are mainly characterized by general irritability, apprehensiveness of impending ills, dizziness, sensations of collapse, together with bodily signs usually associated with fear, such as dyspnœa, palpitations, vaso-motor disturbances, tremors and digestive troubles. Phobias of various kinds are often present. Sleep is frequently disturbed by frightening dreams.

As will be seen, the symptoms closely resemble those of other forms of neurosis, but are always grouped round the main one, which is a feeling of anxiety.

The state is sometimes spoken of as a neurosis as distinct from a psychoneurosis because the anxiety, though of course a psychic symptom, is considered to be of somatic origin, arising, according to the theories of Freud, from inadequate psychic reactions in regard to sexual life. On this hypothesis there is unused psychic energy in the mind of a "free floating" nature which may remain as an indefinite feeling or may become attached to some definite idea. In addition to psychotherapy, any physical symptoms, such as indigestion or constipation, should be carefully treated.

GENERAL SUMMARY AND TREATMENT

A classification of psychoneuroses on the lines indicated above, while useful for working purposes, must not be interpreted too rigidly, since many of the symptoms overlap, and other groups may be added and other divisions made according to the views which different authorities take on the causation of these diseases. Moreover, it is not desirable to insist on labelling every patient with the name of a disease, as symptoms by no means always lend themselves to our provisional classifications. Further, it will be seen from the table on p. 482 that the symptoms which can be classified are to a large extent offshoots from a common root, and it is to that root that we have to look for causation and indications for treatment. Rows* has recently emphasized this view when he suggests that the primary result of excessive emotional reactions is a disturbance of the capacity to perceive, to think, to feel, to judge and to act. This disturbance, he considers, causes a condition of anxiety which, if untreated, will give rise to a sense of dread as to future developments and lay the patient open to receive all kinds of suggestions from without and within. Further developments will result in the appearance of secondary symptoms, the nature of which will depend largely on the circumstances and general mental make-up of the patient, and it is on these secondary symptoms that the classification of symptoms and differentiation into diseases are made.

Treatment of the psychoneuroses consists of such psychotherapeutic methods as seem most applicable to each case, combined with such rest and bodily treatment as may also be required. The main object underlying the different forms of psychotherapy is to "explore" the patient's mind and to find out the origin of his nervous symptoms, so enabling the source of the trouble to be recognized and the ideas to be rearranged with a view to their better adaptation to the events of daily life. In many cases of recent and simple origin a few conversations in which the psychological aspects of the symptoms are carefully considered will be sufficient to enable the patient to recover from his lack of adjustment, but in others the difficulties are much greater and demand a great deal of time and trouble if they are to be overcome.

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* Morison Lectures, *Edin. Med. Journ.*, 1920.

The most elementary form of mental therapeutics is that known as simple **suggestion,** which is used, consciously or unconsciously, by all practitioners. It is not an easy matter to define exactly what is meant by suggestion, but broadly speaking it is the power which some people have of impressing their views on others. It is met with in daily life, and recognized in people whose presence seems to give others confidence. In therapeutics it is shown in acquiring the confidence of patients, in making them feel they are being helped, and in convincing them that their troubles are exaggerated and often without foundation.

By suggestion it is frequently possible to start a beneficial train of thought in a patient's mind that will supersede those that were troubling him. This line of treatment merges into that known as **persuasion,** which, as elaborated by Dubois, consists in explaining to the patient his illogical trains of thought and putting the circumstances before him in a rational light. In this way an attempt is made to rearrange the association of ideas and so to re-educate the mind, but, since the success of the method depends largely on the personality of the physician, it is impossible to separate it entirely from suggestion.

Hypnotism is a method by which the patient is put into a state in which he accepts suggestions more easily than usual. Wingfield* defines hypnosis as " a psychical condition in which suggestions are not only much more easily accepted but are also realized with an intensity much greater than is possible to the normal state." This method has been found useful in curing certain cases of hysterical anæsthesias, contractures and paralyses, and also for recovering lost memories (amnesias), which are often associated with a high degree of emotional disturbance.

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* "An Introduction to the Study of Hypnotism," 2nd ed., 1920.

The method introduced by Freud, known as **psychoanalysis,** is an elaborate and lengthy process of analysing the contents of the patient's mind with the object of getting at the root of the trouble.

Allusion has already been made (p. 484) to the system of psychology whereby Freud supposes that, in certain circumstances, experiences, especially those of a painful nature, may fail to be duly associated, and, being painful to the individual, may gradually become pushed out of the conscious memory and relegated to the sphere of the unconscious mind, where they may still remain active and cause trouble.

The essential point of Freud's method is the attempt to bring such submerged experiences to light and to associate them with conscious experiences, whereby they assume their rightful proportion among the events of the individual's life, and, no longer isolated, cease to be a source of disturbance.

The method by which this is sought to be accomplished consists, as the term psychoanalysis implies, in analysing the patient's mental contents until the root of the evil is discovered.

Originally Freud made use of hypnosis for this purpose, but eventually he discarded it for the more simple method of "free association," which consists essentially in persuading the patient to give out his thoughts indiscriminately without self-criticism and regardless of their seemingly slight importance. By this means the physician picks out the salient points and gradually bores down to the offending source.

As an additional means of arriving at the contents of a patient's mind, Freud has elaborated, at great length, his system of analysing dreams. According to him, a dream represents the fulfilment of some desire which has been repressed in waking life. This essential point is generally so grotesquely clothed that a successful analysis is often a very complicated matter. The subject is too intricate to be dealt with here, and the reader is referred to special works on the subject.

A further and important method of probing a patient's mind is that of "word association," which has been especially elaborated by Jung, and of which some account has already been given (p. 70).

Where the nervous symptoms are due to general toxæmia, such as, for example, might arise from intestinal intoxication, pyorrhœa, or chronic alcoholism, or to a disorder of metabolism such as might be caused by general diseases or modification of some of the internal secretions, it is clearly a mistake to treat the nervous symptoms as constituting the primary complaint, since the only hope of ultimate success lies in curing the disease on which the nervous symptoms have been grafted. On the other hand, there are patients in whom the nervous symptoms have occurred primarily through acute emotional disturbance or more chronic stress, as already intimated. Between these two classes there are those in whom the causation is complicated by the occurrence of emotional disturbance in an already diseased body; and, further, nervous disturbance of primary origin is always, as stated above, accompanied by disturbance of bodily functions, especially those of the alimentary and cardio-vascular systems.

When the cases are regarded from this point of view the reason that so many different forms of treatment are advocated will be readily understood, but success can only be gained by careful adaptation to the requirements of each particular case.

In cases where the nervousness is clearly secondary to some coexisting disease or disorder of metabolism, it is obvious that the treatment must be directed towards the alleviation or removal of the cause, the nervous symptoms being looked upon for the time being as a secondary matter and requiring no form of treatment other than that connected with the general treatment of the disease on which they depend.

Cases of this class will not here be further considered, but attention will be given to those in which the symptoms are primarily due to a failure of the nervous system, and in which palpitation, disorders of the digestion, and other bodily symptoms are secondary to the nerve disturbance and not the cause of it.

Mental treatment.—It is difficult to draw any distinct line between the different methods of psychotherapy, since one often merges into another, and Bernard Hart* has well summed up the matter when he states that all the methods depend upon the use of three main principles, viz. suggestion, persuasion, and analysis, the differences being due to the proportion in which these principles are combined and the technique by which they are applied. In practice, every case must be considered on its merits. In many, a relatively simple "exploration" of the mind is sufficient to clear up the symptoms, while others, especially when the symptoms are of an hysterical nature, can be quickly relieved by suggestion or hypnosis. Yet others may need the longer and more technical process known as psychoanalysis, the success of which, again, depends greatly on the careful selection of suitable cases and the discretion with which the treatment is carried out.

Bodily treatment.—Among the most important of the disordered physical processes which often form the groundwork of mental symptoms are fatigue (mental or physical) and worry.

Everyone is familiar with the modification of mental activities that is apt to occur after a year's work, when power of concentration becomes diminished and things appear out of perspective, so that trifling worries often cause anxiety out of all proportion to their importance. In slight cases of this kind, absence from work and change of scene are generally sufficient to effect a cure, and the worker returns from his annual holiday ready to face the responsibilities of another year. Such cases show the comparative ease with which a cure can often be effected when the disease is taken at an early stage of its development. If, however, the symptoms seem at all severe, it is often a satisfactory plan to combine the holiday with some definite form of treatment, such as can be obtained at one or other of the many good health-resorts at home or abroad. There, while the patient can enjoy his surroundings, he will have part of his day's occupation mapped out for him and his general mental and bodily health will be supervised.

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* *Proc. Roy. Soc. Med.*, 1918.

In more severe cases adequate treatment can only be obtained by a preliminary period of complete isolation from all worries and responsibilities, and for such patients a short time in a nursing home or some similar institution, where psychotherapy can be combined with massage and suitable dieting, is often necessary before a more liberal holiday is undertaken.

In cases where sleeplessness persists in spite of the general treatment, drugs may have to be given; but, broadly speaking, hypnotics should as far as possible be avoided.

Regulation of the bowels is important. The symptoms of neurasthenia are in a considerable proportion of cases associated with constipation. Indeed, some writers consider that neurasthenia is nearly always the result of a toxæmia which arises within the intestines, and which, presumably, is made more intense by constipation. In any case, many of the patients are greatly worried by this condition of things, which has "got on their nerves," and they have usually tried to correct it in a spasmodic manner, which only brings temporary relief.

The choice of purgatives is naturally very large, but must be adapted to the object in view, which is that of restoring regular rhythmical actions. Until regularity is restored, enemata given frequently are often very useful.

Errors of refraction, if present, should be corrected, since a continuous strain of the ocular muscles often leads to irritability and exhaustion.

Suitable drugs in the form of sedatives and tonics, various forms of electrical treatment, and hydrotherapy all have their uses in selected cases.

The last phase in the course of treatment is to prepare the patient for the resumption of occupation.

One of the most critical periods in the course of "functional" nervous affections is that connected with return to work, and the longer the patient has been away the more difficult it is to recommence. This statement is applicable both to those who work with their minds and those who work with their bodies. It is unreasonable to expect a man of the labouring class to do a full day's work at the termination of an ordinary "rest cure," during which he has taken little or no exercise and has almost certainly put on weight. The usual advice to begin with light work is sound enough in itself, but as a rule cannot be taken because there is no light work available; and the fact has to be recognized that it is increasingly difficult for a man to get employment unless he can do his full share of work. Moreover, men of the labouring class, who have been in the habit of going to work mechanically day after day for a number of years, find it difficult on their own initiative to make a fresh start when their routine has been so definitely broken. The result is that the patient, feeling himself unable to do his ordinary work, becomes depressed, concludes that he is not cured, and tends to degenerate physically and mentally into a condition in which he is unable to make any effort to help himself. To remedy this state of things, physical exercises should be prescribed as soon as the preliminary treatment has improved the patient's condition sufficiently for them to be undertaken. The exercises should be given daily for fixed periods, and gradually increased until he is in thoroughly good physical condition and fit to go straight away to his occupation.

On the mental side, encouragement should be given to occupy the mind as soon as there is a definite inclination to do so. At first the patient will often be content to lie in bed for a time and do nothing, but when the mind becomes more active it is a mistake to enforce complete idleness, and some light occupation, such as reading or needlework, should be suggested as soon as the patient feels able to do it. As further progress is made these occupations may be extended and various others devised to suit the intellectuality of the individual and the character of his work.

It must not be forgotten that the performance of physical exercises has a beneficial effect on the mind, not only on account of the sense of well-being produced as the patient gets stronger, but also directly by the performance of voluntary movements, which assists volition and strengthens the coördination between mental processes and voluntary acts.

Towards the latter part of the convalescent stage recreation and occupation should be further extended, and everything possible should be done to enable the patient to readjust himself to his ordinary surroundings.

CHAPTER LIII

NEURALGIA; HEADACHE; MIGRAINE

NEURALGIA

Etiology.—Neuralgia is the term given to recurrent nerve pains which do not depend upon any obvious organic lesion, but it is often used in a more general sense to indicate many other kinds of nerve pain.

The cases may on this basis be divided up into *primary* and *secondary*.

Primary neuralgia, with which is associated no discernible organic lesion, is especially associated with exposure to cold, debility, toxæmias, arterio-sclerosis, eye-strain, and irritation of the peripheral ends of the nerves, e.g. from carious teeth.

Secondary neuralgia often accompanies inflammation, injuries, and degenerations of nerves. Of the last, the pains of tabes and those accompanying and following herpes zoster are good examples.

Symptoms.—Pain is the most prominent symptom; it is generally intermittent, of a sharp, stabbing nature, and sometimes tends to recur with great regularity at certain times in the day or night. The skin over the painful area is often tender to the touch, and in severe cases the nutrition of the hairs suffers, so that they turn white or fall out. Crops of herpes may appear over the painful area.

It is very common to find " painful spots " where the nerve twigs are superficial and can easily be pressed upon.

2 H

Attacks of very acute pain may be accompanied by a reflex spasm of the muscles situated near the painful part. This is especially notable in severe cases of trigeminal neuralgia, and constitutes the condition known as tic douloureux.

Diagnosis.—Whenever possible, it is necessary to distinguish a primary neuralgia from one of secondary origin. Endeavour must therefore be made to exclude the probability of inflammation, pressure, injury, or degeneration as causes.

A line cannot always be drawn sharply between neuralgia and neuritis, but in well-defined cases the intermittence of the pain and the absence of anæsthesia, of muscular wasting, and of any changes in the electrical reactions of the muscles are indicative of neuralgia.

The lightning pains that occur in the course of tabes are apt to be diagnosed as simple neuralgia before other signs become manifest. Gummata and other tumours must also be considered as possible sources of irritation.

Prognosis.—Slight attacks of neuralgia, such as are commonly complained of by people who are feeling "run down," usually disappear when the general health improves.

Severe and persistent pain is, however, often far more difficult to cure, especially when it sets-in in the course of the trigeminal nerve, and in some cases of trigeminal neuralgia no treatment short of removing the Gasserian ganglion may suffice.

No hard-and-fast rules can be given, for the likelihood of recovery in every case can only be gauged after the effects of treatment have been ascertained.

Treatment.—The objects aimed at are—(1) improvement of *general health*, (2) removal of *poisons* such as those of gout or lead, (3) removal of sources of *irritation* of the nerve-endings, (4) administration of such *drugs* as are known to influence the pain, (5) *injection of substances* into the nerve tissues, or *destruction of the nerve* by surgical means.

The methods of attaining the first two objects depend on the application of general principles, which need not here be discussed in detail; they include, chiefly, attention to the digestion and the general hygiene of the patient, and the correction of anæmia if present. The removal of all sources of peripheral irritation is especially important in neuralgia of the trigeminal nerve, which is often the result of eye-strain or decayed teeth; these will again be alluded to in the section on trigeminal neuralgia.

The *drugs* from which relief from neuralgia can often be obtained are numerous, but are not very dependable.

Phenacetin, aspirin, antifebrin, and antipyrin, with preparations of a kindred nature, are among the most popular of these drugs.

Iodides are useful in certain cases where arteriosclerosis and chronic rheumatic processes appear to be the cause of the pain.

Gelsemium sometimes cures the pain of facial neuralgia very successfully, but it is generally necessary to give it in doses of sufficient strength to produce unpleasant physiological effects.

Morphia will, of course, relieve the pain, but it should, needless to say, be reserved for the most occasional use only.

Alkalis in large doses, especially ammonium carbonate and ammonium chloride, are often useful.

Externally, ointments of menthol and other substances that dull the sensibility of the nerve-endings are valuable; and where there are any chronic inflammatory changes round the nerve, massage and galvanism may afford relief.

In cases where the pain does not yield to the ordinary methods of treatment the question of obtaining relief by injections into the nerve may be considered. This method was introduced by Schlösser, and has been elaborated by Brissaud, Sicard, Wilfred Harris and others. It is based on the fact that injection of absolute exclude the presence of organic disease of the cord or meninges, of tumours in the thorax, and of toxæmias of some constitutional condition such as renal disease.

INTERCOSTAL NEURALGIA

Neuralgia of the intercostal nerves is frequently met with. The pain which follows the course of the intercostal nerves is apt to be more continuous than in many other varieties, but is subject to acute exacerbations.

Tender points are generally present (1) close to the vertebral column where the posterior branches come to the surface, (2) in the line of the axilla corresponding to the area supplied by the lateral branches, and (3) where the anterior cutaneous nerves pierce the muscles to reach the surface of the chest.

Intercostal neuralgia often precedes the appearance of herpes, but in these cases it is scarcely correct to speak of the condition as neuralgia, since it has been shown that the herpes is accompanied by hæmorrhagic and inflammatory changes in the posterior root ganglion. The intercostal pains may persist for a long time after the herpetic eruption has subsided.

In making a **diagnosis** the principal conditions from which intercostal neuralgia must be distinguished are (1) pains from irritation or degeneration of nerve roots, as may occur in tabes or tumours of the cord; (2) referred pains from visceral disease; (3) myalgia, in which the pain originates in the muscles; (4) pains from chronic pleurisy.

HEADACHE

To ensure any success in diagnosing and treating headaches it is necessary to take a wide view of their causation, and not to attribute them all to some one special fault, such as an error of refraction or an unsuitable diet.

It is when headache is the main or only complaint that difficulty is most likely to arise, for when it is accompanied by other definite symptoms it is not generally difficult to assign to it its proper place in the disease.

The chief conditions in which headache is likely to appear as the most prominent symptom may be classified as follows:

1. Intracranial disease.
2. Disorders of the urinary system;
3. Disorders of the cardio-vascular system
4. Disorders of the blood.
5. Migraine and toxæmias;
6. Eye-strain.
7. Reflex causes.

Routine **examination** of all cases is necessary if mistakes are to be avoided, for neither the position nor the character of the headache can be relied on as a guide to the cause, nor can a headache which is paroxysmal be safely ascribed offhand to an evanescent cause.

At the onset it is necessary to make sure that the pain is of the nature of "headache" and not of neuralgia, since the causation and treatment of the latter are often very different.

So far as the nervous system is concerned, unless the history specially points in that direction, it will generally be sufficient in the first place to examine the optic discs, the reaction of the pupils, the ocular movements, and the reflexes—bearing in mind the possibility of the existence of intracranial growths, cerebral syphilis, tabes, and general paralysis of the insane—and to follow up the investigation in greater detail if any suspicious signs are elicited. The results of the examination of the urine will indicate the presence or absence of disease of the kidney.

In the cardio-vascular system the tension of the arteries must be noted, for a persistent low or high blood-pressure will sometimes cause headache, even though there be no obvious disease of the kidneys.

Valvular lesions of the heart sometimes cause headache, either through anæmia, such as may be associated with aortic regurgitation, or congestion following dilatation of the right ventricle. There is not often much danger of overlooking the cause in either of these conditions, but congestion may, of course, arise from conditions other than those associated with valvular disease.

After the presence of obvious disease of the larger systems has been excluded, the eyes and the sinuses may be thought of, and when thinking of the sinuses it is well to remember that nasal obstruction may be a cause.

Errors of refraction and accommodation, due to astigmatism and hypermetropia, are among the commonest causes of headache, and are often associated with seemingly typical attacks of migraine. Simple myopia does not usually give rise to headaches.

The migraine and toxæmic group of headaches is a large one. The characteristics of migraine are referred to in the next section, and, under the hypothesis of toxæmias, diet and intestinal action have to be considered, and must be regulated according to the necessities of the individual case.

Headaches sometimes appear to occur reflexly, as the result of sensory impulses from the ears, eyes, and other channels. Head has shown that many headaches of this kind are "referred" pains depending on diseases of the various viscera.

As already stated, the locality and character of a headache are not usually reliable guides either to the position or to the nature of the lesion. Exceptions are, of course, sometimes seen, perhaps chiefly in the case of the headaches of intracranial syphilis, where the painful area is apt to be localized and tender, in addition to which there may be the further characteristic of nocturnal exacerbations.

In most cases of cerebral tumour the headache produced by the general rise of intracranial pressure does not admit of differentiation from that produced at the seat of the growth, though, taken in conjunction with other symptoms, the signs are sometimes very suggestive, as when, for instance, a dull pain occurs over one half of the occipital region and at the same time there is reason to suspect the pressure of a tumour in the cerebellum.

Treatment.—Variations in the blood supply—congestion, hyperæmia, or anæmia—though produced in many different ways, seem to be at the bottom of most headaches, and it is by altering the character of the circulation in some way or other that relief is often obtained.

It goes without saying that, whenever possible, the cause should be removed; but when no definite cause can be found it is necessary to try to ensure the healthy working of all those organs through which the headache is likely to arise, and particularly the eyes, the alimentary canal, and the kidneys. The eyes must be examined, and vision corrected if necessary. The bowels must be kept open by suitable purgatives, and the kidneys must be washed out by simple diuretics, of which water in plentiful amount is one of the best. At the same time that the output is being looked to, attention must be paid to the intake, and a plain diet, containing a minimum amount of purin bodies, prescribed.

For empirical treatment aspirin, phenacetin, antipyrin, and antifebrin are among the most useful drugs. Calcium salts have been used with benefit in cases in which headaches seem to be associated with a deficient coagulative power of the blood. It is said that the subjects who suffer from headaches of this kind are generally of a "lymphatic type," i.e. they have a heavy, listless expression, with pale face and puffy eyelids, and their headaches are generally felt most in the mornings. The lactate of calcium, which is more palatable than the chloride, may be given in doses of 10 to 20 grains three times a day. This drug is said to be also very useful for relieving the morning headaches that follow the taking of too much alcohol on the previous night. Among other drugs that are useful are salicylates, bromides, caffeine, and cannabis indica.

Where there is persistent high arterial tension, vasodilators, such as nitro-glycerin and trinitrin, are sometimes given. They have only a transitory effect, as a rule, and more general methods to relieve the pressure should be employed.

Excess of food, by disturbing the chemical processes of digestion, can undoubtedly cause headaches. In the proteid diets it is probably the purins that are harmful, but it has been shown by Hare that headaches of a similar nature can also arise from taking too much carbohydrate food.

MIGRAINE (HEMICRANIA)

Migraine is a paroxysmal neurosis characterized by headache and sickness, which are often preceded by visual symptoms.

Etiology.—More women suffer than men, and there is a strong tendency for the disease to run in families. Occasionally other neuroses, such as hysteria and epilepsy, are prominent in the relatives of patients, but in a large number of instances the patient and his family are quite free from any other special form of nervous weakness.

In epileptics the fits sometimes alternate with attacks of migraine, and it is generally supposed that the pathology of migraine is analogous to that of epilepsy, inasmuch as both appear to depend upon a disturbance of the cells of the cerebral cortex ; but while it is reasonable to assume that the greater explosions of epilepsy would sometimes include the lesser of migraine, clinically there is, at any rate, a very wide interval between the average patient with migraine and the sufferer from epilepsy, and patients who are subject to typical attacks of migraine fortunately show no special tendency to become epileptics, though it is undoubtedly easy enough to find plenty of instances where both have occurred and, perhaps, alternated in the same person.

Gout and gouty tendencies have been noted in association with migraine, and it has been considered by some that uric acid is the actual cause of migraine, but of this there is not sufficient proof. In many people the first attacks date from childhood, and appear to become more frequent towards the middle decades of life; in others a few isolated attacks occur from time to time. In those who are the subjects of migraine, attacks are often induced by physical or mental fatigue, indigestion, alcohol, and work requiring close application of the eyes.

Errors of refraction are notably a cause of the attacks.

General summary of causation.—It is necessary to insist on a broad view being taken of the causation of migraine if success is to be obtained in its treatment. On the whole it appears that some people are more prone than others to attacks of migraine, and that, in them, various causes, such as fatigue, errors of refraction, and dietetic indiscretions, are capable of inducing an attack, when they would be harmless to other individuals.

The nature of the condition that determines the tendency to migraine is not definitely known, but it is reasonable to suppose that it is a poison generated either in the alimentary canal or in the tissues themselves during the chemical processes taking place in the latter, and that it acts upon the brain cells either directly or indirectly by the changes produced in the cerebral circulation.

Vaso-motor spasm, followed later by dilatation, has always been a plausible hypothesis, and is supported by the pallor of the face and extremities which is often observed at the beginning of an attack and is followed later by flushing; on the other hand, experiments have not shown the presence of vaso-constrictor nerves in the cerebral vessels. It has also been suggested that the attacks may be due to periodical enlargement of the pituitary body.

Symptoms.—The symptoms may be conveniently divided into three groups:

1. Ocular.
2. Headache.
3. Sickness.

1. **Ocular symptoms.**—In the majority of cases the eye symptoms are the first to occur, but occasionally they are preceded by sensations which pass up a limb, like, but of longer duration than, the aura of epilepsy, and are often accompanied by feelings of giddiness. The visual symptoms take various forms, of which the chief are flashes of light in front of the eyes, scotomata, coloured spectra, hemianopia, and transient general dimness of vision.

Flashes of bright light may start the attack and then spread out gradually into coloured spectra of zig-zag shape.

The scotoma is usually nearly in the middle of the field of vision, and shows itself to the patient by the existence of a black patch in whatever he looks at, the edges of the patch being zig-zagged, from which the condition has received the names of "fortification spectra" and "teichopsia." The coloured spectra may show themselves as zig-zag ribands of different colours.

Hemianopia is of the simple or homonymous variety, i.e. there is a corresponding loss of half-vision in each eye, so that the sight is lost towards the right or the left, as the case may be. This often comes on very quickly, and the patient finds he is only able to see one half of the objects he looks at. At other times there is a general dimness of vision, and complete blindness for a time has occasionally been observed.

2. **Headache.**—Following on the eye symptoms comes the headache. This often begins at one spot in the region of the right or left temple, and thence spreads over one half (hemicrania) or the whole of the head. At other times the painful spot on the one side is followed by a pain over the corresponding area of the other side before the headache becomes general.

3. **Sickness.**—The headache generally increases in intensity for a time, and is succeeded by a sensation of nausea, which may be followed by retching and vomiting; the headache, in its turn, fades away, and so the attack passes off.

Additional sensory symptoms which sometimes occur are numbness and tingling or even temporary weakness of a limb, and occasionally a passing aphasia may be met with.

It must, of course, be clearly understood that all these symptoms are not present in every case of migraine. In many the headache, with perhaps a certain amount of nausea, is the most prominent symptom, and in these the nature of the disease is often revealed by the periodicity of the attacks.

Duration.—The duration of an attack varies from a few hours to a day or more. Some people are incapacitated for about two days, but, as a rule, recovery takes place after a night's rest.

Prognosis and treatment.—Cases in which a tendency to the attacks is only slight can often be cured by getting rid of some form of irritation which excites the attacks. It is in this way that many patients obtain relief by wearing suitable glasses.

In some the most careful correction of errors of refraction and the removal of any other obvious source of irritation make but little difference, and in others again no error of refraction of any importance exists.

Diet.—The diet should be light, and, although there is no proof that uric acid is the cause of the attacks, many cases certainly seem to benefit by abstaining almost entirely from meat.

In severe cases it is worth while to go farther and try what a strict vegetarian diet will do.

Early hours should be kept, fatigue avoided, and the bowels kept regular.

For warding off attacks, bromides and salicylates are among the most useful drugs, and act better in combination than when given separately. When the patients usually feel the symptoms on first awaking, the medicine should be taken the last thing at night. In other cases it may be given twice or three times a day for a time, and then gradually reduced.

Very little can be done to cut short an attack beyond resting and applying cold to the head.

Such drugs as phenacetin, antipyrin, and caffeine, if taken at the very beginning, sometimes afford relief; but once the symptoms have gained a fair hold, drugs seem to have little effect—probably the stomach is not in a suitable condition to absorb them.

The methods of treating migraine may be summed up as follows :—

During attacks.

1. Rest in a darkened room.
2. Cold to head.
3. Phenacetin Dg 6 (gr. x), aspirin Dg 6 (gr. x), antipyrin Dg 6 (gr. x), or antifebrin Dg 2 (gr. iii).

Between attacks.

1. Avoidance of undue fatigue and excitement.
2. Correction of errors of refraction or any other source of peripheral irritation.
3. Regulation of diet. Digestible food, with restriction of meat.
4. Prescription : e.g. potassii bromidi Dg 6–10 (gr. x–xv), sodii salicylatis Dg 6–10 (gr. x–xv), spiritus chloroformi Dl 12 (♏xx), aquam ad Ml 30 (℥i)—taken three times a day, or a larger dose every night.

OPHTHALMOPLEGIC MIGRAINE

This is a somewhat uncommon condition, in which the periodic headaches are accompanied by temporary paralysis of the third nerve with its attendant symptoms of ptosis, strabismus, and fixed dilated pupil.

CHAPTER LIV

VERTIGO

Etiology.—A sensation of giddiness may accompany a great many morbid conditions, of which the most important are disorders of the *ears*, *eyes*, *heart*, *stomach*, and *kidneys ; intracranial growths*, especially those connected with the cerebellum ; and *epilepsy*. Alcoholism, excessive smoking, and other toxæmic conditions may also cause vertigo.

AURAL VERTIGO

Disorders of the ear are one of the most common causes of giddiness.

So far as the ear is concerned, the sense of equilibrium depends on the pressure of the endolymph and on the state of the nerve terminals in the semicircular canals, which through the vestibular nerve form communications with the cerebellum. Anything, therefore, that varies the pressure or alters the irritability of the nerves may upset the normal equilibrium and cause vertigo.

The pressure of the endolymph within the semicircular canals may be modified by chronic inflammatory changes in the middle ear, which may be of gouty, senile, or syphilitic origin, whereby the intensity of the vibrations conducted through the chain of ossicles is increased. Wax, which is one of the commonest causes of vertigo, probably produces its effects in the same way, for a solid piece, if pressing against the drum of the ear, serves as an additional conductor, and causes the intensity of the vibrations reaching the labyrinthine nerves to be increased. In other instances an undue irritability of the nerve fibres or their centres is the cause of the vertigo. The labyrinth itself may be the seat of injuries, of effusions, or of inflammations.

The symptoms of aural vertigo are also sometimes associated with high arterial tension and Bright's disease, quite apart from the giddiness, which may be a symptom of uræmia.

Vertigo of labyrinthine origin is known as Menière's disease, but the latter term is often used to include other forms of aural vertigo.

Symptoms.—In a typical case of aural vertigo the patient suddenly feels *giddy*, and either has a sensation of movement or actually moves; at other times, apparent movement of surrounding objects is the chief thing noticed. In severe cases the patient may suddenly be thrown to the ground or may only save himself by holding on to some neighbouring support.

After the giddiness has lasted a variable time it is often succeeded by *nausea* and *vomiting*. Occasionally temporary *diplopia* and *nystagmus* have been noticed, the latter being due to the disturbance of coördination which exists between the movements of the head and eyes (*see* p. 92).

Tinnitus often immediately precedes a paroxysm and sometimes also persists between the attacks; some degree of *deafness* is frequently to be found on one or other side. The nature of the deafness can be tested by a tuning-fork. The vibrations from a tuning-fork placed on the forehead are ordinarily heard in both ears equally, but if the conducting apparatus of the ear is impaired they are heard loudest on the side of the deaf ear.

On the other hand, when the deafness is of nerve origin the vibrations of the tuning-fork placed on the forehead are heard best on the sound side.

Further, in nerve deafness, after the vibrations of the tuning-fork placed on the mastoid have been lost they can generally still be heard when the instrument is held opposite the meatus.

Diagnosis.—Attacks of *vertigo* associated with *deafness* and *tinnitus* are the main points to be established. And further evidence can be obtained by carrying out the tests for labyrinthine disease described at p. 92.

The vertigo of *epilepsy* is sometimes difficult to differentiate, but if the attacks are repeated frequently, some positive signs of epilepsy, in the shape of loss of consciousness, convulsions, biting the tongue, and involuntary micturition, are almost sure to be obtained. The most difficult cases to decide are those in which the patient actually falls, for in those circumstances there is naturally some confusion of mind which is not easy to differentiate from the loss of senses due to epilepsy. If a history of loss of consciousness can be established, the condition is almost certainly one of epilepsy. It is doubtful if vertigo of anything like the severity described above ever arises from *gastric disorders*, though, of course, "dizziness" and "giddiness" are often complained of by patients who suffer from indigestion.

Tumours of the brain, especially those of the *cerebellum*, are frequently accompanied by giddiness, but other signs of intracranial pressure are generally present.

It is necessary to remember that occasionally the giddiness of *hysterical patients* may simulate that of Menière's disease, but, needless to say, it is necessary to be very cautious before ascribing a case to this cause.

Prognosis.—In aural vertigo only very rarely is such an acute attack as Menière recorded associated with changes likely to endanger life. Possibilities of improvement must, of course, largely depend on the anatomical changes that have occurred. In a large number of cases partial relief can be obtained, and the giddiness often gets less by the time the morbid changes are sufficiently advanced to cause complete deafness.

Treatment.—The ear must be carefully examined, and treated if necessary. If any wax is present it must be removed. In some cases this alone is sufficient to effect a cure. If the giddiness cannot be accounted for in this way, recourse must be had to drugs, and of these the bromides, in 15- to 20-grain doses twice or three times a day, are probably the most efficacious. Rheumatic and gouty cases may derive benefit from salicylates and iodides, which may be given in combination with the bromides. When the symptoms are due to disease of the middle ear, small doses of pilocarpine nitrate ($\frac{1}{100}$ to $\frac{1}{50}$ gr.) may be beneficial. Quinine sulphate is a drug that was recommended by Charcot, who considered it necessary to give it in large doses, but many authors believe small doses to be equally efficacious. Counter-irritation in the shape of a blister over the mastoid is sometimes useful. If these measures bring no definite relief, it may be well to put the patient to bed for a week or two, for complete rest is often a most useful therapeutic measure. If the arterial tension is too high, purgatives and careful dieting are frequently beneficial. If all else fails and the attacks continue to be unbearable, the question of obliterating the semicircular canals may be entertained.

Treatment of vertigo from other causes.—All forms of vertigo are generally relieved to some extent by taking bromides, and any further course of treatment must depend upon the cause, whether it be heart, stomach, arterial degeneration, cerebral tumour, or ocular palsy.

CHAPTER LV

VASO-MOTOR NEUROSES

DISORDERED functions of the vaso-motor nerves are sometimes the cause of various unpleasant symptoms, most of which can be grouped under one of the following headings:

Raynaud's disease.
Angioneurotic œdema.
Erythromelalgia.
Acroparæsthesia.

RAYNAUD'S DISEASE

The **symptoms** of Raynaud's disease may be divided into three stages:

(*a*) *Local "syncope,"* in which the fingers, toes, and perhaps the nose and ears, become pale and "dead."

(*b*) *Local "asphyxia,"* in which the initial pallor is followed by congestion, so that the part becomes blue in colour.

(*c*) *Gangrene*, in which small parts of the asphyxiated areas necrose and separate, leaving little scars.

A large number of patients whose symptoms come under this heading never experience all these three stages. Many people habitually suffer from "dead" fingers, especially in cold weather or after putting the hands into cold water, and in some of these the stage of "local" asphyxia is also experienced; necrosis occurs in comparatively few.

Etiology.—Spasms of the vessels of the extremities appear to be the cause of the disease, and the spasm is usually attributed to a "vaso-motor neurosis." What causes the neurosis is uncertain, but probably, at any rate in some cases, it is the result of a toxæmia. Symptoms of a similar nature to those described above may be found associated with syringomyelia, and they are also especially prevalent during the early stages of some cases of rheumatoid arthritis.

Vaso-motor affections of the kidney resulting in paroxysmal hæmoglobinuria are sometimes associated with Raynaud's disease, and also closely allied to it clinically are the ordinary chilblains of winter weather.

Friction and electricity are the most useful forms of **treatment**, and in the case of the latter the constant current, applied daily, is the most efficacious method of administering it. The hand and the positive pole should be placed in a bowl of warm salt and water, and the negative pole should be applied to the upper part of the limb.

Drugs are of little direct value, but those that have a general tonic action may be given, and beneficial results are sometimes obtained from nitro-glycerin, sodium nitrite, and other vaso-dilators.

The symptoms occur in paroxysms and are always worse in cold weather; hence care should be taken to wear warm gloves and socks.

ANGIONEUROTIC ŒDEMA

Œdema occurs in the skin and sometimes in the mucous membranes of different areas of the body. At one time the face may be the seat of the swelling, and at another the limbs or the trunk. The œdema lasts a variable time and then disappears. It gives rise to some discomfort and irritation, and if present in the mouth and pharynx may make swallowing uncomfortable; œdema of the glottis may occur and give rise to symptoms that are dangerous to life.

Closely allied to angioneurotic œdema clinically are the urticarias which sometimes follow the ingestion of shell-fish, pork, and some other foods.

The history of the varying amount of the swelling generally helps in distinguishing its nature, but the urine should always be carefully examined so as to exclude the possibility of Bright's disease being the cause of the œdema.

Sometimes more than one member of a family suffers from angioneurotic œdema.

Treatment.—It is not easy to cure angioneurotic œdema. Attention should be directed towards the alimentary canal, and careful dieting, with abstention from foods that usually give rise to urticaria, may be tried. With the same object in view, intestinal antiseptics may be given.

Nitro-glycerin and calcium lactate are among the drugs that have been recommended.

It is important to maintain a high standard of general health.

ERYTHROMELALGIA

This condition usually occurs in the distal parts of the arms or legs. The arteries dilate, the skin becomes hot and red, and there is great pain. After a time the redness of the skin disappears, and is replaced by a bluish colour. The intensity of the symptoms is increased by holding the affected limb in a dependent position, and also by warmth or exercise. When arterial degeneration is present, intermittent claudication and cramps may be associated with this disease.

Symptoms of erythromelalgia may be met with in association with various organic diseases of the nervous system, e.g. multiple neuritis and tabes, and with arteriosclerosis; at other times no sign of organic changes can be found, and the disease is then attributed to a vasomotor neurosis. The symptoms are generally relieved by keeping the limb high and by cool applications to the affected part. Massage and electricity may also be useful, as they are in Raynaud's disease.

ACROPARÆSTHESIA OF VASO-MOTOR ORIGIN

Here the patient complains of tinglings, pins and needles, and numbness of the extremities (usually of the arms). In most of the cases the symptoms come on in the night. The patient goes to bed and falls asleep, but after a time wakes up with tingling and numbness, which may be sufficiently severe to prevent any more sleep being obtained. On getting up and using the limbs the sensations usually pass off, but they are very apt to recur during the day if the patient rests for any length of time, especially if near a fire so that the limbs become hot.

The symptoms are more commonly found in women than in men, and especially in those who are engaged a good deal in washing. The use of alcohol also seems to favour their onset.

The women sufferers are often overworked and generally debilitated, and some of them are at the menopause period of life.

Tingling, numbness, and "crawling sensations," accompanied in some instances by some anæsthesia, are frequently met with over the area of the thigh, which derives its cutaneous nerve supply from the external cutaneous nerve, and to this condition the term *meralgia paræsthetica* has been applied. In the majority of cases no lesion of the nerve can be found, and we have to fall back on a vaso-motor neurosis as the cause.

Diagnosis.—Before concluding that the symptoms of acroparæsthesia are due to a functional disturbance, care must be taken to exclude the presence of peripheral neuritis, locomotor ataxy, or other organic conditions with which symptoms of a similar nature may be associated.

The **prognosis** of these cases is, on the whole, good if the patient can be placed amidst favourable surroundings, but recovery is generally slow and the relapses are frequent.

Treatment.—Iron, nux vomica, and other tonics are helpful, with a view to restoring the health, but a thorough change is best of all if it can be procured.

Electricity and massage are both useful.

It is difficult to prescribe anything which will be certain to relieve the symptoms at the time, but the bromides seem to afford as much relief as any drug. If sleep be much interfered with, other hypnotics may be required.

Vaso-motor Neuroses of General Distribution

These neuroses have already been alluded to in the chapter on the Autonomic System (p. 46). They include such vaso-motor symptoms as flushing, shivering, palpitation, and dyspnœa, many of which are associated with neurasthenia and with the period of the menopause.

The bromides, nux vomica, strychnine, and the vasodilators are among the drugs that are likely to give relief.

CHAPTER LVI

EXOPHTHALMIC GOITRE (GRAVES'S DISEASE, BASEDOW'S DISEASE)

Etiology.—Exophthalmic goitre is far more commonly seen in women than in men. In the majority of the cases the symptoms first appear between the ages of 15 and 40, and it is rare to find the disease arising in elderly people.

In some instances nothing that seems to have definitely led up to the origin of the disease can be traced, but in a considerable proportion of the cases fright, shock, and long-continued emotional strain of the nervous system appear to bear a decided relation to the onset of the symptoms. Auto-intoxication from the alimentary canal or other sources also appears to be a cause.

Pathology.—The view that meets with most favour at the present day is that which attributes the symptoms to hypersecretion or some other modification of the functions of the thyroid, and possibly also the parathyroid glands. The influence which the secretion of the thyroid gland normally exerts on the general metabolism of the body can be fully appreciated from the ill effects which arise on its removal; and if disappearance of the gland can produce such far-reaching results, it is not difficult to understand how modifications in the composition or amount of the secretion can produce peculiar effects of another nature. This view receives support from the frequency with which changes in the thyroid gland are found in exophthalmic goitre, and it is also a noticeable feature that many of the symptoms are almost the exact opposite of those which occur in myxœdema, where the secretion of the gland has disappeared. Moreover, excessive doses of thyroid gland are liable to cause symptoms similar to many of those associated with exophthalmic goitre; and, lastly, relief from the symptoms of exophthalmic goitre is often obtained by ligation of the arteries or by partial extirpation of the gland.

In some instances an enlarged thymus gland has been found, and changes of an inconstant and somewhat indefinite character have been noted in the ganglia of the cervical sympathetic and in the medulla.

Though the idea that lesions of the autonomic system are primarily responsible for the disease has for the most part been abandoned, a large number of the symptoms are undoubtedly produced through the agency of this system, and the close relationship that is known to exist between the internal secretions and the autonomic system makes it apparent how such a secondary affection can arise. The sequel of events, then, would appear to be: (1) Emotional disturbance or an infection; (2) over-activity of the thyroid gland; (3) over-activity of the autonomic system and general increase of metabolism.

Symptoms.—The onset in most cases is gradual; only occasionally is it rapid.

The order in which the signs appear differs, and there is also great variation in their number and intensity, but the principal ones are four, viz. enlargement of the thyroid gland, tachycardia, exophthalmos, and tremors.

Enlargement of the thyroid gland.—This sign is present at some period or another in very nearly all cases, and it may precede other symptoms by a long time. Like the other manifestations of this disease, the enlargement is not only very various in different cases, but is apt to vary from time to time in the same case. In some patients it is so slight as to be almost unnoticeable, while in others the gland is symmetrically or asymmetrically enlarged to a great size. The vascularity of the enlarged gland is usually increased, the pulsation of the larger arteries running into it can be seen and felt, and not infrequently a thrill and a murmur over the tumour can also be detected. Pressure of the gland on the trachea may cause dyspnœa.

Tachycardia.—Rapidity of the pulse-rate is a characteristic symptom; from 100 to 120 beats per minute are commonly met with, and frequently the number is very much higher.

Pulsation of the heart is generally more obvious than usual, not infrequently there is some dilatation, and a systolic murmur can often be heard at the apex. In long-standing cases there may be hypertrophy, and eventually signs of cardiac degeneration may be found.

Tracings by means of the electro-cardiograph show that the rapidity of the heart is due to an exaggeration of the physiological beat, and is produced at the expense of the diastole.

The blood-pressure in a typical uncomplicated case is low owing to dilatation of the peripheral vessels, the latter being one of the most frequent features of the disease, as evinced by the patients generally having a moist and flushed skin.

The interesting observation has been made that after the thyroid gland has been operated upon in Graves's disease the blood-pressure is very apt to rise. The systolic pressure may even be doubled, thus throwing a great strain on the heart, and so in some cases possibly accounting for sudden deaths that have occurred (Strickland Goodall).

Exophthalmos.—This is not an invariable symptom. When it is present the prominence of the eyeballs gives the striking appearance of fright to the face. Protrusion of one eye before the other sometimes occurs.

The cause of the exophthalmos has been much debated. It has been attributed to various conditions, e.g. increased vascularity, increase of fatty tissue at the back of the eyeball, and contraction of Müller's unstriated muscle, which runs across the back of the orbit.

Other ocular symptoms which can frequently be observed are: *Von Graefe's sign*, in which the dropping of the eyelids is delayed when the eyes are directed downwards; *Stellwag's sign*, in which there is retraction of the upper lid due to spasm of the unstriated muscle fibres; *Moebius's sign*, in which there is difficulty in converging the eyes to view near objects; and *Joffroy's sign*, in which the forehead is not wrinkled on looking upwards while the head is flexed on the chest.

Tremors.—A fine rhythmical tremor of the hands, which can often be better felt than seen, is met with in most of the cases.

The remainder of the symptoms, which are numerous, may be briefly summarized under the headings of the different systems to which they are attached.

Nervous system.—Patients suffering with exophthalmic goitre are apt to be nervous, excitable, and very easily upset. They often sleep badly, and they may develop symptoms of hysteria and other slight forms of mental derangement. In a few cases the mental changes are profound enough to constitute a state of insanity.

Respiratory system.—In the more severe cases attacks of dyspnœa may occur, apart from those due to mechanical pressure of the thyroid upon the trachea.

Alimentary system.—The appetite is variable, as also is the power of digestion. Vomiting and diarrhœa, more commonly the latter, are both met with.

Renal system.—The urine is generally plentiful in amount, and often contains a high percentage of urea. Glycosuria and traces of albumin are found in some cases.

Skin.—The skin is usually moist, and perspiration is easily induced. Brown pigmentation is frequently noticed.

The **general nutrition** is poor; loss of weight is a characteristic feature, and the patients are sometimes very emaciated. In women uterine disturbances are often met with.

Diagnosis.—There is no difficulty in diagnosing a case in which the main symptoms are well developed. There is, however, always a possibility of overlooking the nature of the disease when general symptoms, such as nervousness and loss of weight, are the most prominent features.

Prognosis.—Improvement, which is usually a very slow process, occurs in a considerable number of cases. In some the symptoms entirely subside, but in the majority some traces of exophthalmos still remain, as also frequently do the nervous and excitable temperaments.

A fatal termination from the direct effects of the disease is distinctly uncommon, but it occasionally happens that the disease runs an acute course and, the patient becoming delirious, death takes place with all the symptoms of profound toxæmia. In a few cases, where the functions of the thyroid gland are eventually destroyed, the symptoms of myxœdema have supervened.

Treatment.—Among the drugs which are of most service, arsenic, belladonna, bromides, iron, and digitalis may be mentioned. Arsenic, chiefly in the form of liquor arsenicalis, is one of the most popular for general use; bromides and belladonna are both serviceable on account of their sedative effects. Iron acts as a general tonic, and digitalis is frequently useful for steadying the heart. When the enlargement of the thyroid is accompanied by much pulsation, comfort is often obtained by placing an ice-bag on it.

Applications of electricity over the course of the cervical sympathetic and over the thyroid gland sometimes seem to produce beneficial effects. The interrupted and constant currents may both be used.

Preparations of the various glands, e.g. thyroid, thymus, and suprarenal, have been tried, but with no distinctly good results. One would, indeed, expect preparations of thyroid gland to be harmful if the hypothesis which ascribes the disease to hypersecretion be correct, but, as a matter of fact, cases have been recorded in which benefit appears to have been obtained from their use. Such results are not necessarily to be taken as invalidating the hypothesis of disordered action of the thyroid gland, for it may be that in some cases the modification of the secretion is one of quality rather than of quantity. A preparation known as rodagen (prepared from the milk of goats which have had their thyroids removed) has been recommended.

Administration of preparations of the thymus gland has also sometimes appeared to be followed by relief of symptoms, but there is no general consensus of opinion in favour of the habitual use of drugs of this class.

Such measures as are likely to improve the general health should be recommended. Rest, sometimes under the Weir Mitchell system, is important, as also are quiet surroundings and general tranquillity of mind.

Application of X-rays to the enlarged thyroid has been tried, and in some cases satisfactory results have been reported.

Surgical treatment is directed towards reducing the activity of the thyroid either by partial removal of the gland or by ligature of one or more of its arteries. It is an operation of considerable severity and one which by no means always results in a cure, for which reasons it should only be performed after due consideration of all the circumstances of each case. Strickland Goodall, who has made some careful observations on the subject, considers that one of the most important benefits obtained by early operation is relief of tachycardia, thus avoiding the danger of dilatation and permanent cardiac weakness, which are both apt to occur when the tachycardia has continued for a long time. Goodall also points out that the rise of blood-pressure which, as already stated, is apt to follow the operation is a source of danger when the heart is debilitated, and necessitates the patient being carefully prepared before the operation is undertaken.

CHAPTER LVII

TETANUS (TRISMUS, LOCKJAW); HYDROPHOBIA

TETANUS

Etiology.—Tetanus is an acute infective disease resulting from the entrance into the body of the tetanus bacillus.

The tetanus bacillus, first described by Nicolaier, is of a drumstick shape and flourishes chiefly in the soil, and from this it is in most cases introduced into the body through punctured or lacerated wounds. Infants are liable to be infected through the umbilical wound (tetanus neonatorum), and parturient women through the placental surface (puerperal tetanus).

Pathology.—The tetanus bacilli remain in the neighbourhood of the wound, and cause symptoms by diffusion of the toxins to which they give rise. These toxins have a special affinity for nervous tissue, by which they appear to be "fixed" after the manner of certain stains.

After death, minute hæmorrhages can be seen in different parts of the central nervous system, which throughout is found to be intensely congested.

Definite changes can often be distinguished in the cell bodies of the brain and cord, the most characteristic of which are swelling of the cell bodies and modifications of the staining properties of the chromatophile granules.*

Symptoms.—After the patient has become infected there is a period varying in duration from two to three days to a month before the symptoms appear. The first intimation of anything wrong is generally a sensation of stiffness in the muscles of the neck, which is soon followed by spasms in the muscles of the jaw and face, by which the jaw becomes locked and the features distorted to produce the "risus sardonicus." Superadded to these tonic contractions are spasms of the muscles of the trunk and limbs, which occur with great frequency and with such intensity as to cause opisthotonos and other contortions of the body. The slightest peripheral stimulation, such as from a loud noise, a draught of air, or an attempt to move, is apt to bring on a spasm.

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* Alexander G. R. Foulerton and Campbell Thomson, *Trans. Roy. Med. Chir. Soc.*, lxxxiii.

Spasms of the respiratory and laryngeal muscles give rise to great distress and difficulty in breathing, and pain produced by the generalized cramps is also a prominent feature.

The frequency of the spasms depends upon the intensity of the infection. The temperature may be raised slightly or not at all during the earlier stages of the disease, but there may be hyperpyrexia towards the end. The mind usually remains clear, and death takes place from exhaustion and failure of respiration.

Diagnosis.—The mode of onset, characterized by rigidity of the muscles of the neck, jaw, and face, when following a traceable cause of infection, seldom leaves room for much doubt concerning the nature of the disease.

Hydrophobia is the disease in which the symptoms most closely resemble those of tetanus, but in hydrophobia the "lockjaw" is absent, and the spasms are especially concentrated on the muscles connected with respiration and swallowing, so that attempts to drink are at once followed by an attack. There is in hydrophobia also the likelihood of a history of the possibility of infection having occurred from a bite of a dog or some other animal.

Poisoning from *strychnine* gives rise to spasms which resemble those of tetanus, but there is more complete flaccidity of the muscles in the intervals between the spasms, and trismus is absent. Moreover, the onset is likely to be far more sudden.

In *tetany* the distribution and characters of the contractions in the hands and feet usually obviate any possibility of confusion, even when the spasms invade the muscles of the trunk.

Prognosis.—A patient who is suffering from tetanus is always in a dangerous condition, and the mortality is said to be more than 80 per cent.

The longer the incubation period the better is the outlook; and when the spasms have set in, the intensity of the infection and the gravity of the case can be to some extent gauged by the strength and frequency with which the convulsions occur.

Treatment.—The site at which infection has taken place should be cleansed and disinfected. This may influence the course of the disease, but, as already stated, the symptoms appear to be due to the diffusion of toxins, and therefore they, unfortunately, do not necessarily subside with the removal of the bacilli.

Treatment by *antitoxin* gives the best chance of recovery, and should be carried out whenever practicable. The earlier treatment is commenced the greater are the chances of success, and whenever there is any reason to suspect the presence of tetanus infection, the antitoxin should be given as a *prophylactic* measure, with a view of neutralizing the virulence of the toxin before fixation in the nerve tissues has taken place. As a prophylactic measure the dose should be from 1,000 to 1,500 units of antitoxin. For curative purposes the doses are much larger—10,000 to 20,000 units or more according to the severity of the symptoms. The antitoxin is best given by subcutaneous or intramuscular injection; there is no evidence to show that intrathecal or intravenous administration has any advantage.

The patient should be isolated in a darkened room, and should as far as possible be protected from all forms of stimulation which are liable to intensify the strength of the spasms.

Drugs must be used to produce sedative effects, and for this purpose bromides, morphia, cannabis indica, and chloral will naturally suggest themselves. When the convulsive attacks are very severe, inhalation of chloroform may be followed by some relief.

Nutrition must be maintained, and if necessary the patient must be fed by a nasal tube.

Periods of dyspnœa may be tided over by performance of artificial respiration.

HYDROPHOBIA (RABIES)

Etiology.—Hydrophobia is transmitted to man by the saliva of animals, such as dogs, foxes, and wolves, which are suffering from rabies.

Pathology.—After death the principal alterations found in the central nervous system are hyperæmia, minute hæmorrhages, and extravasation of leucocytes into the perivascular tissues.

Symptoms.—The incubation period is very variable, but the usual interval which precedes the onset of symptoms is about six weeks, though cases have occasionally been recorded in which twelve or even eighteen months had elapsed before the signs of infection made their appearance.

Vague sensations referred to the seat of the wound, which in the meantime may have healed perfectly, often first lead to an idea that something is wrong, and this suspicion is soon confirmed by the advent of convulsive attacks somewhat similar to those of tetanus, but differing from them in the fact that the intensity of the spasms is especially concentrated on the muscles concerned in respiration and swallowing. It is due to the spasms of the latter that the act of drinking becomes so difficult, for they occur whenever the patient attempts to take fluid, and even the sight of water is often sufficient to bring them on.

These convulsive seizures may spread to other parts, and are brought on by the slightest stimulation, such as a sudden noise, or a draught of air impinging on the body; when the muscles of respiration are implicated there occur intense and painful attacks of dyspnœa, which then become a prominent feature of the case.

The temperature is raised, and wandering of the mind with hallucinations and delusions is frequently present.

Death occurs from exhaustion brought about by the spasms, and it may be preceded by a paralysis beginning in the lower limbs, similar to that which occurs in animals.

Diagnosis.—The main differences between the symptoms of hydrophobia and *tetanus* have already been mentioned (p. 543).

The convulsions arising from poisoning by *strychnine* are not concentrated in the muscles concerned in swallowing, as they are in hydrophobia. *Tetany* can scarcely give rise to any serious difficulty, but *hysteria* has occasionally been known to simulate the symptoms of hydrophobia.

Prognosis.—The danger of a hydrophobia occurring after the bite of an animal suffering from rabies is greater when the patient is bitten on unprotected parts of the body, such as the face and hands, than when the animal's teeth have passed through the clothes, the latter acting as a barrier to the entrance of the poison. About 15 per cent. of the patients bitten by dogs suffering from rabies eventually develop symptoms of hydrophobia.

Treatment.—A recent bite should be freely cauterized as soon as possible, and, if none of the usual methods are at hand, rough-and-ready means, such as the application of some heated instrument to the wound, can usually be devised. Diffusion of the poison may be also retarded by applying a ligature tightly round the limb and encouraging free bleeding.

Once the poison has been absorbed, the only method of treatment by which it can be hoped to stave off the disease is that of inoculation with the attenuated virus as devised by Pasteur. The length of the incubation period makes it possible to render the patient immune by the inoculation of attenuated virus of gradually increasing strength, which is prepared from the spinal cords of rabbits that have been infected with the disease. By this means the mortality, which in untreated cases is very high, has been greatly reduced, and, provided the case is taken in time and treated thoroughly, there is every chance of the toxin being neutralized before it has had time to cause any symptoms.

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